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A REVIEW OF OUR PRESENT KNOWLEDGE OF PNEUMOCONIOSIS, BASED UPON ROENTGENOLOGIC STUDIES, WITH NOTES ON THE PATHOLOGY OF THE CONDITION*

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THE word "pneumoconiosis" is of Greek origin, and implies a condition of the lungs arising from the inhalation of excessive quantities of dust. The fact that damage to the lungs may be wrought by the inhalation of certain dusts has been recognized for many centuries, but the exact type of dust or dusts responsible for the pulmonary changes and the nature of the pathological processes induced are of comparatively recent knowledge. Systematic roentgen-ray studies, painstaking pathological investigations following autopsies and abundant animal experimentation throughout the world during the past ten or twelve years have led to a very exact understanding of this most interesting condition.

America is far behind many other countries in the methodical study of pneumoconiosis and the practice of preventive measures. Comparatively few systematic investigations have been carried out. In 1916, one of us, in conjunction with Miller and Landis, instituted clinical and roentgenological investigations of the lungs of workers in a large number of dusty industries, and our preliminary report was published in 1918.[1] These studies have been carried on intermittently ever since.[2] The United States Public Health Service and the Bureau of Mines have been carrying out a routine study of dusty occupations, especially mining, for even a longer period. Their first report including roentgen-ray observations was made by Lanza and Childs[3] in 1917. Jarvis and his coworkers[9–14] have contributed much valuable data from their studies of the granite cutters of Barre, Vermont. The experimental work of Gardner,[22,23] Permar[65] and others has added much to our knowledge of the pathological processes involved in pneumoconiosis. We can be justly proud of Miller[27–32] for the wonderfully clear knowledge he has imparted to us of lung histology, and particularly of the lymphatic system, upon which to base our conception of pulmonary pathology. Notwithstanding all these and many other American contributions, the amount of work accomplished in the study of pneumoconiosis in this country cannot compare with the extent to which the condition has been studied abroad, especially in South Africa, Australia and Great Britain. Naturally, in the latter country, especially, the concentration of dusty industries has been a definite aid to all observations. In those countries not only

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have the intensely interesting phases of the subject stimulated investigation, but also the preservation of the health of the workers has demanded a thorough understanding of the condition and the adoption of adequate protective measures. In South Africa and Australia, particularly, the governments have been forced to take a hand in the control of the dust hazards through medical commissions. Probably the geographical extent of the United States and the scattering of dusty industries have been in a large measure responsible for the lack of more concentrated effort in investigation and the difficulty of control.

NOMENCLATURE

If one's knowledge of the subject were derived entirely from the literature he would be easily led to the conclusion that of all dusts silica alone is the one responsible for serious pulmonary changes. A great part of the literature on the subject has emanated from Great Britain, South Africa and Australia where a large amount of hard rock mining is done and the damage to the workers' lungs has been, beyond doubt, due entirely to silica. In those countries the condition is almost universally spoken of as "silicosis" rather than "pneumoconiosis." It is probably true that all serious damage is due to silica. Were it true that silica is the only dust capable of producing lung fibrosis it would seem proper to drop the nonspecific word "pneumoconiosis" from our nomenclature, and also the more specific subsidiary terms anthracosis, siderosis, chalicosis, (pottery dust), etc. Indeed, it was our own conviction at one time that silicosis was the only term that should be used to designate the condition of pulmonary dust fibrosis, but the more experience one gains in roentgenologic investigations of various dusty industries, the more he is inclined to retain and employ pneumoconiosis as a general descriptive term and silicosis and possibly the others to designate the condition arising from special industries. Anthracosis, siderosis and chalicosis imply occupation only, as the active agent in all of them is silica. In coal mining the condition (anthracosis) is somewhat different in that silica is the active fibrosing agent and the coal dust is relatively harmless or produces quite different effects. The term "miners' phthisis" has been used in South Africa and elsewhere and is said to have dated from the time when little or no distinction was drawn between pneumoconiosis and tuberculosis. In South Africa the term legally implies silicosis.[18]

ROENTGEN-RAY DIAGNOSIS

It is generally conceded that the roentgenologic examination is the easiest and most reliable method of studying the effects of dust inhalation in the living subject, and yet our knowledge of pneumoconiosis from this point of view is comparatively recent. The literature is already replete with articles on this phase of the subject, although most of the writers have merely outlined roentgenographic appearances in general, reported a few isolated cases or have dealt extensively with appearances noted in workers in a single industry. Purdy,[41] of Australia, in 1922 collected 103 separate contributions to the subject of pneumoconiosis.

The first important contribution on the roentgenologic aspect of the condition in this country was made by Lanza and Childs[3] in 1917, and the roentgenographic classification they instituted has been generally followed here. In the same year, Boardman[4] published a review of the literature to date. The most comprehensive American roentgenologic studies bearing on a single industry have been made by Jarvis.[10–14] Probably the earliest collective roentgenologic studies on a large scale were made by the South African Miners' Phthisis Commission, and their published report in 1916 on investigations of the Rand Miners[18] still stands as a classic on the subject of pneumoconiosis from the standpoint of etiology, pathology, symptomatology and roentgenologic features. Anyone interested in the subject should not fail to read this most enlightening report.

While there is a great similarity in the roentgenographic appearances of the chests of individuals exposed to any of the harmful dusts which contain high percentages of silica, and one who studies the workers

exposed to silica inhalation in a single industry is not apt to become biased in his opinions, except possibly in minor details, such an observer continuously along one line has exceptional opportunities offered for studying his cases more intimately. He can watch the slow progress of the condition and observe retrogressions in the abnormal appearances during periods of intermission from work and their reappearances on resumption of occupation. Jarvis has added more than any other American observer in this way, and by means of serial roentgenography, to our knowledge of the changes in the lungs over a considerable period of time. Our collective knowledge of the subject must depend upon the work of many such observers in single industries.

Roentgenologic interpretations of any morbid process in the lungs must be based upon a thorough knowledge of the anatomy and histology of the lungs and the pathological processes peculiar to the infecting or etiological agent. One must acquire the habit of thinking in terms of pathology and rendering deductions upon a pathological basis because he is interpreting the shadows of disease processes. Lung histology has been most clearly demonstrated by Miller,[27–32] and every roentgenologist should be thoroughly familiar with his graphic and concise descriptions. There is still much diversity of opinion in regard to the many pathological details of pneumoconiosis and it has seemed wise to review the investigations and conclusions of various writers before further consideration of the roentgenologic aspect of the condition.

DUSTS

While all dusts when inhaled in excessive quantities may be harmful, only a few can be regarded as dangerous. From the standpoint of their dangerous properties they may be divided into three general groups: (1) Those which are dangerous because of their poisonous action, as lead, arsenic, mercury, etc.; (2) those which cause only irritation in the respiratory tract, resulting in such conditions as chronic bronchitis or asthma; and (3) those which tend to produce pulmonary fibrosis, and may thereby predispose to respiratory infections, especially tuberculosis and pneumonia. It is with the last group only, and with silica especially that we are mainly concerned in the study of pneumoconiosis. The most important pathological observations have been made in connection with the effects of free silica, SiO_2, in a finely divided state. Silicates are comparatively innocuous. Probably several other dusts besides free silica produce fibrosis, but no others do so to anything like the extent that silica does. Purdy[41] in a recent review of 103 contributions on the subject of silicosis found it to be the consensus of opinion that while most dusts may have some injurious effects, silica is the most dangerous of all. Landis[42] states that any inorganic dust may cause lung trouble, varying in degree from that produced by almost pure silica to that of the relatively innocuous cement dust.

It is probable that only by continued roentgen-ray investigations can we reach any definite conclusions as to the relative effects of inorganic dusts other than silica. Very little convincing animal experimentation has been done with other dusts than silica. Other dusts can produce fibrosis in man only after a period of many years, and certainly the observations on animals over a few months will not show changes comparable to those which require twenty to forty years in the human.

The important factors in the production of lung fibrosis are: (1) The time at work or exposure to the dust; (2) the quantity of dust present; (3) the quality of dust with special reference to silica, and (4) the size of the particles. It was formerly believed that sharp points and edges were the dangerous factors in a dust but it is now known that this is not the case. Sharp pointed or edged particles may be irritating to the respiratory passages, but upon this basis alone they play no part in lung fibrosis.

A considerable percentage of dust particles are too large to be taken up into the lungs, and are either expectorated, swallowed or possibly dissolved. Watt, Irvine, Johnson and Steuart[18] state that over 95 per cent of mine dusts floating in the air

are less than 12 microns in diameter. They quote McCrae[25] who demonstrated that most of the particles found in the ash of silicotic lungs were less than one micron, and very few were found microscopically to be over 8.5 microns. Moir[26] is quoted as determining that 93 per cent of the particles extracted by acid digestion from silicotic lungs were less than 2.5 microns. Middleton[33] calls attention to the fact that silica dust in the lungs can be seen only by polarized light under the microscope. He also reminds us that the naked eye appearance of a very dusty atmosphere is no criterion as to the damage-producing properties of the dust without a knowledge of the size of the particles. Because of the small size of the dangerous particles he does not believe that respirators could stop all of them, and if made to exclude all fine dust they would embarrass respiration.

Large percentages of free silica are encountered in the dusts developed in hard rock mining, especially in gold, copper, zinc, lead, silver, nickel and cobalt mines. Anthracite coal mining is more likely to produce pneumoconiosis than bituminous mining. In mines the dust is confined and settles if not removed by ventilation, and accumulates for long periods of time. In hard rock mining the most dangerous occupation is usually that of the driller. Watt, Irvine, Johnson and Steuart[18] state that of all the workers in South African mines rock drillers are the most affected. Investigations made in 1911 showed that of all such men who had worked four and one-half years, over 50 per cent were found to be affected to some degree clinically, whereas of all other workers for the same period only 29 per cent were affected. After eight and one-half years, 75 per cent of drillers and 39 per cent of all others were affected. After fifteen years the proportions were 100 and 50 per cent respectively. In South Africa dry drilling is said to be prohibited by law. Our own experiences in interpreting for the United States Bureau of Mines the roentgenograms of chests of workers in mines throughout the country have made it possible for us usually to pick out those of drillers or driller's helpers provided we know the length of time of employment of the individuals. Blasting and crushing also produce large quantities of fine dust to which many kinds of workers may be exposed.

Granite cutting, for which a pneumatic tool is used, is a very dusty occupation, and the dust contains a high percentage of free silica. Many other occupations are more or less hazardous because of the prevalence of a high percentage of free silica in the dust developed in the industry, such as potters, sand paper makers, tool grinders, metal polishers and grinders, brick makers, especially firebrick, and occupations in which sand blasts are used.* Individuals living in sandy localities where dust storms are prevalent frequently show fibrotic lung changes. We have noticed this in some farmers, and Steuart[19] speaks of transport riders, mail train attendants and farmers of South Africa showing evidences of pneumoconiosis.

Not all hard rock mining and quarrying is dangerous. Workers in pure limestone or marble may acquire pneumoconiosis, but it never reaches the advanced stage found in the workers in silica rocks. Mavrogordato[36] states that in one hard rock quarry in South Africa no free silica was found in the rock and no cases of definite silicotic fibrosis were found among the workers. In animal experimentation with the dust from this quarry the dust cells were found to retain the particles for a long time. There was not so much tendency for lymph block, and while fibrotic changes did occur eventually, they were very mild as compared with those produced by silica. Similar observations have been made in many other localities, and it is probable that the changes are similar to those observed in the majority of our coal miners.

PREVENTIVE MEASURES

It has been the experience of those who have investigated the causes of silicosis, and especially in South Africa, Australia and England, that much of the

* Our attention has been called recently to the likelihood of a hazard in the occupation of sand blasting buildings in cities. Although the men work in the open, there is ample opportunity afforded for the inhalation of rather large quantities of silica.

serious damage to the lungs can be prevented. Investigations leading to the protection of workers in the countries mentioned were instituted many years ago. Kerr[61] states that in 1902 the Sewage Works Ventilation Board investigated the workers in sandstone tunnels around Sidney, Australia, where the dust contained 81 to 94 per cent of free silica. In 1904, the Royal Commission on Ventilation and Sanitation of Mines in Western Australia advocated large exhaust fans. Most of the investigations at the times mentioned were instituted because of the prevalence of tuberculosis among the workers. Kerr[61] refers to the investigations in Australia of Simmons, who found that six times more deaths occurred from tuberculosis among the Blendigo miners than among the other population. Many other similar boards have followed in the work of prevention, notably the Miners' Phthisis Prevention Committee of South Africa.[18] Steuart[19] states that this Bureau was established at Johannesburg under government auspices in 1916, and was composed of a chairman, nine full time medical officers and a roentgenographer (Steuart). They examine all applicants for mine work in scheduled mines, and all the miners every six months, and also all applicants for compensation. Independent clinical and roentgenological examinations are made and then submitted to the Board.

Precautionary measures are of two general types, those directed against the production of pneumoconiosis and toward the prevention of tuberculosis in the workers who have developed advanced silicosis. Dry drilling is the first danger and probably the most important one, and, as previously stated, this is prohibited by law in South Africa,[18] at least. Blasting releases enormous quantities of fine dust, and the particles which are sufficiently small to cause damage are the ones which stay suspended in the air for the longest time. All blasting is recommended at the ends of shifts, to be followed by water spraying. Kerr[61] states that the Technical Commission at Broken Hill, Australia, requires that one-half hour elapse after blasting before any workers are allowed in the vicinity. Dust stirred up by walking, transport or shovelling can also be kept down by water. Water is regarded as the most reliable precautionary agent, although ventilation is very important to remove unlaid dust. Rock crushing is also dangerously dusty, but can be controlled. Kerr[61] states that in 1922 the Technical Commission at Broken Hill, instituted in 1920, following an upheaval of mine employees, expressed the opinion that with certain precautions in mining the amount of dust could be so reduced that advanced pneumoconiosis would not occur. This statement is, of course, open to serious question as applied to mining in general. It refers to one general type of mining. It is possible that the use of water may not be practicable in all mining industries. Finlayson[67] states that the enforcement of adequate ventilation, adequate use of water, blasting at ends of shifts and preventing re-entrance until blasting dust has settled have radically changed the hazards of the Broken Hill district since the commission started work in 1920.

In dusty factories, such as asbestos and cement works, cleanliness and ventilation are essential, although the actual danger in these two industries seems to be comparatively slight. In our investigations in a number of potteries we found carelessness easily corrected by cleanliness and suitable receptacles into which the moulders could drop waste clay. The kiln rooms would be more difficult to render perfectly safe.

The worker who has developed advanced silicosis, especially if the progress has been rapid, is predisposed to tuberculosis. In 1921, the Technical Commission of Broken Hill[61] took steps to exclude all tuberculous and silicotic workers from the mines, in both surface and underground workings, to provide employment elsewhere for the silicotics and to compensate the tuberculous and disabled silicotic individuals. The Commission insisted upon periodic examinations of all workers. This was but following out the practice of many other similar bodies such as the Miners' Phthisis Prevention Committee of South Africa.[18]

In the work of all commissions such as those mentioned, the roentgen-ray examination plays the most important rôle in determining the effects of dust upon the lungs. It is likely that much more roentgenographic study will be made of workers in dusty occupations in this country in the future, and it behooves us to be familiar with the subject. Textbooks and standard works on roentgen diagnosis have either omitted the subject altogether, or have given it scant mention.[6,7,8]

PATHOLOGY

The pathological features of pneumoconiosis are best arranged for consideration under the following headings: (1) The entrance of dust; (2) the "dust cell;" (3) the entrance of the "dust cell" into the lymphatic system; (4) the subsequent conversion of the "dust cell" into fibrotic tissue; (5) the elimination of dust; and, (6) the predisposition of the fibrotic lung to respiratory infections. Most pathological studies and experimental work have been carried out in connection with silica.

The Entrance of Dust. A large part of inhaled dust is removed from inspired air before it reaches the alveoli of the lungs because of the natural protective mechanism of the respiratory tract. Much of it is removed in the nose and pharynx, where it is caught by the secretions and blown or coughed out or swallowed. Farther down, the mucus bathed walls of the trachea and bronchi catch the particles, and the cilia down as far as they exist, by their movements propel them upward so that they can be expectorated or swallowed. The very fine dust which gets beyond the ciliated epithelium reaches the alveoli, and the pathology of pneumoconiosis begins.

Watt, Irvine, Johnson and Steuart[18] state that in their experience mouth breathers are more apt to develop silicosis than nose breathers. White,[58] who has studied pneumoconiosis in men and horses believes that man is more liable to the condition than the horse because he is largely a mouth breather while the horse is a nose breather. Jarvis,[11] on the other hand, found that for the same exposure periods mouth breathers among the Barre granite workers showed fewer changes than nose breathers. He also found[13] that those whose upper respiratory tracts were most free of excessive lymphoid deposits and other defects or conditions, such as deviated septa, large turbinates and atrophic rhinitis, lasted longer without disabling lung damage.

The "Dust Cell." The presence of irritating dust particles in the pulmonary alveoli sets up a catarrhal process with the proliferation of certain cells. At least some of these are phagocytic and incorporate the smallest of the particles. They have been given the name of "dust cells." The process from now on is an unusual and a most interesting one. There has been much controversy over the exact origin and identity of the "dust cell." It has been variously identified as an alveolar epithelial cell, a mononuclear leucocyte or an endothelial cell. Willis[21] states that some observers have thought that the dust particles passed directly between the epithelial cells, or pierced them, but that most observers believe that the particles enter the lung tissue by cellular or phagocytic activity. He gives references to all authors whose views he quotes or comments upon. From his own experiments on animals he concludes that practically all dust in the lungs is intracellular, and that the "dust cells" are transported through the lymphatics.

Mavrogordato[36] has made an exhaustive study of the subject, both pathologically and experimentally. He believes that the "dust cells" are of endothelial origin from the walls of the capillaries and lymphatics, and that they become wandering cells. Experimentally he has traced them from the incorporation of dust particles in the alveoli to the formation of fibrous tissue. Silica dust, at least, seems to provoke their proliferation. There is a very intimate relation between the alveolar epithelium and the endothelium of the capillaries. The alveoli are, of course, lined by epithelium, which is either adjacent to a single layer of endothelial cells of the blood or lymphatic capillaries, or the epithelial cells are interrupted by some of the endothelial cells. Mavrogordato

thinks this arrangement favors the latter as the "dust cells" or phagocytes for dust. If his deductions are correct that the "dust cell" later changes to fibrous tissue, this would certainly seem to exclude the epithelial cell as the specific phagocyte in question, although it is known that it may become phagocytic. His experiments have failed to show that the polymorphonuclear leucocyte takes up either silica or coal dust, although some authors state that they have found dust particles in these cells. Mavrogordato shows that the essential difference at this stage between dust and microorganisms such as the tubercle bacillus is that the former mobilizes the tissue mononuclears alone and there is no congestion of blood vessels or migration of polymorphonuclear cells to deal with irritating agents. In his experimental intravascular injections of fine dust to further prove which cells were "dust cells," he found that the particles were taken up mostly by the endothelial cells of the blood vessels.

Permar[65] quotes Haythorn[63] as stating in 1913 that he believed the "dust cell" was an endothelial cell probably originating from blood or lymph vessel linings. He also quotes Sewall[64] as believing, in 1919, the "dust cell" to be an alveolar epithelial cell. His own experiments, published in 1924,[65] have led him to believe that the "dust cell" arises entirely from capillary endothelium. Into rabbits showing a mild degree of anthracosis he injected intratracheally a saline suspension of finely ground carmine powder. The carmine was phagocyted rapidly and was all intracellular in twenty-four hours by large mononuclear cells which had arisen by proliferation of capillary endothelium. Some cells were found still *in situ;* others could be traced from capillary walls to alveolar spaces. Subsequently, many remained in the alveoli, but some re-migrated into the tissues, gained the terminal lymphatics near the alveolar ducts, and some were traced passing slowly toward the hilum lymph nodes, where the first arrived in twenty-four hours after the injection. Incidentally, Permar believes that the disposal of the tubercle bacillus may occur in the same way. He found that the mononuclear cells of the alveolar exudate in pneumococcus infection were derived from the same source.

The evidence submitted would seem to favor the endothelial origin of the "dust cell."

The Entrance of the Dust Cell into the Lymphatic System. No doubt many of the dust laden cells are expelled through the respiratory passages, but a large number reach the lymphatic system of the lungs. Many observers have traced the dust cell from its entrance onward. Gardner[23] in his observations on animals exposed to granite dust found the following movements of dust cells after discontinuance of dust exposure. The cells gather in the alveoli located along the alveolar ducts and move toward the lymphoid tissue at the distal ends of these passages, thus, one by one, entering the first outpost of the lymphatic drainage system. This lymphoid tissue then undergoes hypertrophy. Cells then move to the next larger lymphoid deposits at the bifurcations of the bronchioles and bronchi, and eventually reach the tracheobronchial lymph nodes. If the lymphatics become choked by a continued dust inhalation, fibrosis ensues, and the dust can no longer be removed. From then on a progressive fibrosis results. So far, we believe that these processes explain very well the first stage changes in pneumoconiosis as shown by the roentgenogram, and described later under roentgenographic appearances.

Middleton[33] states that dust cells may be found in clumps between the alveolar walls in the perivascular lymphatics, which are occluded thereby, and in the more proximal lymph nodes.

According to Mavrogordato[36] silica dust, unlike coal dust, remains intracellular, and the dust cells tend to aggregate into groups which he calls pseudotubercles. It is in these bodies that fibrosis begins. They resemble tubercles except in the absence of giant cells and the periphery of lymphocytes and polymorphonuclear cells, and in subsequent fibrosis instead of caseation. Jarvis[13] very aptly calls attention to the fact that the course of dust particles from the bronchus to the lung parenchyma is essentially the same as that of the

tubercle bacillus, but that subsequent events differ.

Mavrogordato[36] further states that silica seems to possess the property of protecting the dust cells from autolysis and lymph digestion, so that they can accumulate and block the lymphatics. In the case of other dusts, like coal, the cells are destroyed and do not block the lymphatics. He believes that silica particles are slightly soluble in alkaline tissue juices, and that an alkaline silicate is precipitated, which preserves the cells.

Conversion of Cells into Fibrous Tissue. The next step following the aggregation of dust cells in the lymphoid deposits and the blocking of the lymphatics is the formation of fibrous tissue. The trend of thought of many observers and experimenters is that the dust cell becomes a fibroblast. Mavrogordato[36] has caused excessive fibrosis in the peritoneal cavity by dusting it with sterile silica. Gye and Purdy[45,46,47] state that silica is insoluble in water and all acids except hydrofluoric, but that it can be dissolved in alkalies. The circulation of silica in nature depends upon the formation of a hydrated colloidal silica, which is a poisonous substance. A guinea pig was quickly killed by a 30 mg. dose. In sublethal doses, its greatest action after intravenous injection was upon the vascular capillary endothelium, and large numbers of these cells got into the circulation. Small daily intravenous doses were followed in a few months by marked fibrosis of the liver, enlargement of the spleen and interstitial changes in the kidneys. Gye and Kettler[48] experimentally proved that colloidal silica was a cell poison. They believe that the fibrosis caused by finely divided silica is due to the slow formation of hydrated colloidal silica, which acts as a cell poison.

Fibrosis is the essential pathological process in pneumoconiosis, and upon it and lymph block depend the characteristic roentgenologic features. It is quite agreed that the process of fibrosis begins in and around the various lymphoid deposits where the dust cells lodge after being taken up in the lymphatic system from the ductuli alveolares. Most observers believe that the presence of silica induces a connective tissue cellular proliferation such as is produced anywhere as a reaction to an irritant. Mavrogordato[36] and others seem to regard the endothelial dust cell as the original fibroblast. It seems probable that as the dust cells die or become fibrous tissue the dust particles, and also pigment particles like coal, if not dissolved, become extracellular and lie within the fibrous tissue. The above description would apply to the beginning of a fine and practically microscopic nodular fibrosis along the courses of the lymphatics arising in the primary lobules and extending centrally along the pulmonary arterial branches, pulmonary venous radicles and bronchi. This process continues until the fibrosis in and around the lymphoid deposits becomes visible as a uniformly scattered nodular fibrosis in which the individual nodules cast shadows on the roentgenogram and depict the second stage of pneumoconiosis. But let us go back again to where the microscopic nodular fibrosis is forming. Along with this another process is instituted. The lymphatic channels become obstructed, there is more clogging with cells and a more diffuse fibrosis begins in the interstitial lung tissue. The interalveolar tissue and the interlobular septa become thickened because they contain clogged lymphatics. At first the lymph flow remains fairly active toward the hilum and the dust cells are carried all the way to the hilum and the tracheobronchial lymph nodes. These enlarge and become fibrotic. Many cells are caught on the way hilumward in the lymphatics which extend centrally, as just stated, and cause fibrosis and clogging along the bronchial tree and produce the thickening of the trunk shadows and linear markings which, together with the enlarged hilum shadows, give the characteristic roentgenographic evidences of the first stage of the condition. The thickening of the interlobular septa, just referred to, may be so marked as to aid in this appearance, or it may not become evident until the nodules enlarge to visible size in the second stage. As a rule the nodular fibrosis holds back until a well-marked first stage becomes evident, but not always in the rapidly

developing silicosis due to inhaling of enormous amounts of fine high percentage silica dust.

As certain sets of lymphatics become clogged, the burden of lymph flow is thrown on others, which in turn, become similarly affected. Naturally, as lymph block toward the hilum becomes marked, there is a tendency for a back flow toward the subpleural lymphatics, wherein similar changes are instituted, which result in fibrous nodules, thickened pleura, adhesions, and even, rarely, effusions may occur. Thickened pleura is found in a large percentage of advanced silicotic cases. As an evidence of the by-passing of the lymph stream we have seen dust pigment demonstrated by Dr. A. J. Smith in the diaphragm where the visceral and parietal pleurae have become adherent.

In the case of nonfibrosing or weakly fibrosing dusts and very moderate silica inhalation, the lymph flow is maintained hilumward, and the individual may not, in the course of thirty or forty or even fifty years, progress past the first stage, or possibly may show evidences of the second stage of nodular fibrosis in only those portions of the lungs where the greater part of the inspired air goes,—around the central portions,—while the remaining portions will remain clear. But if the inhalation of silica is in greater quantities, more pronounced and widespread changes occur. The small fibrotic nodules increase in size and coalesce to form larger and macroscopic nodules, from pinhead to pea size, so characteristic of the roentgenologic second stage. The interstitial fibrosis also continues in the interalveolar tissue and interlobular septa, and may encroach upon the alveoli, bronchioles and vessels.

There are, as will be noted, two simultaneous processes going on. The nodular change is the striking one so far, but the way is being paved for advanced and more serious conditions,—a generalized fibrosis and incapacity. If the dust inhalation remains moderate in degree, the generalized process takes place very slowly, and the condition may remain one of an essentially discrete nodular fibrosis, at least for a long time, and not pass this extent in thirty or forty years. But if the dust is more intense, the generalized process proceeds more rapidly, and diffuse fibrosis very soon becomes the prevalent condition. This we recognize as the third stage. Such a rapidly progressing diffuse fibrosis probably explains why we frequently find extensive third stage appearances without much evidence of those of the second stage and still less of the first stage.

Steuart[19] and others state that the generalized fibrosis obliterates the alveoli and compresses the bronchioles and capillaries and renders the lung unable to properly oxygenate the blood, thus causing dyspnea. This may be true, to some extent at least, although we have not taken this view of the cause of the extreme dyspnea and lack of expansion observed during the roentgenoscopic study of a large number of cases of advanced pneumoconiosis. We have always regarded the lungs in these individuals as being inelastic and incapable of expansion by respiratory effort, because of the excessive generalized fibrosis, fibrous band and pleural adhesions limiting the diaphragmatic movement and even the costal expansion. These individuals simply cannot take even an ordinary breath. Moreover, many of the lungs present a distinctly emphysematous appearance. Possibly both causes are operative in extreme dyspnea.

The third or advanced stage of fibrosis may take on at least three distinct appearances, or all three may be present in the same case,—a diffuse fibrosis with nodules still present, larger nodules due to coalescence of small ones, or large irregular areas of consolidation due to massive fibrosis. These changes will be described further in connection with roentgen-ray appearances.

Elimination of Dust and Recovery. It seems to be a well-established fact that individuals who have not progressed far into the third stage of silicosis may show decided clinical and roentgenographic improvement upon cessation of their dusty occupations. Jarvis[11] has observed this many times among the Barre granite cutters when their work has been inter-

rupted for a considerable period of time because of strikes or for other reasons. This has been explained experimentally and by pathological study. Watt, Irvine, Johnson and Steuart[18] state that the lung is capable of freeing itself of dust to a certain extent, even after the dust is in the lymphatic system and lymphoid deposits, by a reversal of the process of entrance, provided new dust is not entering too fast. Mavrogordato[36] states that it has been determined experimentally that six months are required between the time of exposure to silica dust and the appearance of evidence of fibrosis. He also states that silica dust has been found in the sputum of men some time after they have suspended work. We have been informed (Dr. F. V. Meriwether, U. S. Bureau of Mines) that a coal miner who stops work may have black sputum for two to four years afterwards. Certainly these and many other observations would seem to indicate that silicosis may be arrested or even regress. Mavrogordato thinks that if an individual continues to grow worse after cessation of work, some infection, especially tuberculosis, is indicated.

Nicholson[50] mentions as one of the three ways by which the respiratory tract may rid itself of dust that the dust cells may either carry the particles within reach of the cilia for expectoration or through the alveolar walls into the lymph stream. Mavrogordato[36] states that the dust cells have two ways of exit,—by expectoration or travelling in the blood or the lymph stream out of the lungs.

While it seems evident that the dust cells which have not progressed to the development of fibrous tissue may leave the lungs, and in this way decrease cell deposits at the lymphoid centers and relieve lymph block, will actual fibrosis disappear?

Predisposition to Infections and Coincident Tuberculosis. All authors who have made any extensive study of advanced rapidly progressing silicosis are in accord in stating that the silicotic lung is predisposed to intercurrent respiratory infections, especially tuberculosis, and to a less extent, pneumonia. Watt, Irvine, Johnson and Steuart[18] state that pneumonia is longer in clearing up because of obstructed lymph drainage. Both irritation of the respiratory tract and fibrosis are the chief factors in the predisposition to tuberculosis. Latent tuberculosis is more likely to become active. Mavrogordato[36] believes that the tuberculous process spreads rapidly in the silicotic lung because it invades the thin walled new blood vessels which have formed as a result of the cutting off of the original ones by the fibrosis of the perivascular lymphatics and the new vessels have not developed normal defenses. While silicosis has been somewhat controlled so that it develops more slowly, tuberculosis still seems to be a serious problem.

Gye and Kettler[48] found experimentally that injections into mice of silicic acid and suspensions of tubercle bacilli in normal saline solution caused a more severe reaction than injections of organisms alone, which, in reality, produced very little reaction. A central zone of coagulation was filled with tubercle bacilli, which migrated into the surrounding tissues through the agency of phagocytes, and set up other foci of infection. They concluded that the association of tuberculosis and silicosis in man was due (1) to the destructive action of silica on the cells, causing necrosis, in which the tubercle bacilli could multiply; and (2) to the disorganization of lymphatic drainage of the lungs. Purdy[41] quotes Collis[40] as suggesting that pure silica is readily combined in colloidal compounds which poison the cells and render them vulnerable to tubercle bacilli.

Landis[42] states that in potters when tuberculosis is superimposed on pneumoconiosis it is usually nontoxic, with only slight constitutional manifestations, and easily handled by rest. This aspect he ascribes to the fibrosis which limits the spread of the disease. He thinks the presence of marked fibrosis would seem to argue in favor of rendering the lungs less susceptible to the tubercle bacillus. This applies to potters and to coal miners. Tuberculosis is more apt to develop late in potters. In the quickly silicoting lungs of other occupations the same changes

take place in two to eight years which take thirty to forty years to develop in potters. In the rapid cases the lungs are acutely inflamed and are a fertile field for tuberculosis.

UNUSUAL COMPLICATIONS

We have observed one case of phrenic nerve paralysis in connection with silicotic lungs of the third stage. We believe that this was due to the effect of the fibrosis upon the nerve. The patient, a male aged fifty-eight, was referred from the service of Dr. Stengel in the University Hospital for an examination of the chest and gastrointestinal tract because of a large mass in the right upper abdomen, vomiting after a full meal and a loss of 90 pounds in weight during six months. He had a rather profuse mucopurulent expectoration and gave a history of having been a tool grinder for seven or eight years and later a farm laborer. Examination of the chest (Fig. 1) showed a very advanced third stage pneumoconiosis, with numerous large areas of massive fibrosis in both lungs. We have been unable to determine whether a coincident tuberculosis was proven or not, as the history of the case was lost. Diaphragmatic excursion was very limited. The left diaphragm was very high but heavy in outline, suggesting an acquired rather than a congenital eventration. On the basis of this appearance we interpreted phrenic nerve paralysis probably resulting from the massive fibrosis. Incidentally the examination of the gastrointestinal tract was practically negative except in showing the usual cascade type of the partly filled stomach characteristic of eventration or left phrenic nerve paralysis.

Hall[20] in an article on tumors suggests that irritants and evidences of inflammatory processes may predispose to primary lung and bronchial tumors, and mentions pneumoconiosis as a possible predisposing cause. We have had occasion to examine many hundreds of chests showing all stages of this condition and have found no instances of proven primary neoplasm. In two instances appearances were somewhat suggestive of a growth, but this was not confirmed clinically or by subsequent events. A third case showed a primary sarcoma of a rib. We have encountered mediastinal evidences of Hodgkin's disease in several individuals with pneumoconiosis, but the diagnosis has always been established by biopsy from glands removed from an accessible location like the neck, and the occurrence of the Hodgkin's disease has undoubtedly been purely a coincidence.

Pleural complications are so commonly met with that they cannot be regarded as unusual complications, and are discussed elsewhere.

RACIAL STATISTICS

According to Jarvis[11] race seems to materially influence the developmental period of silicosis and the severity of the process for a given time of exposure. Italians seem to suffer the least, and then in order are Americans, English, Spaniards, Scotch, Swedes, Norwegians, Danes, French and Irish. He states[9] that among the Barre granite cutters the Italian workers usually escape the severe forms of the condition, the percentage of third stage cases among them being one-half that of other races. No explanation is forthcoming. Watt, Irvine, Johnson and Steuart[18] state that the native South African miners appeared to be more susceptible to silicosis than those from overseas, with the possible exception of men drawn from the races of Southern Europe. It would appear to us that in determining these facts one would have to consider carefully the previous occupation, duration of exposure and the exact character of dust to which the imported miner was previously exposed.

PIGMENTATION OF LUNGS

Nearly every one is familiar, through autopsy experience or hear-say, with the darkly pigmented lungs of coal miners and of individuals exposed to large quantities of some other dusts. While such pigmentation is frequently, or perhaps usually, associated with silicosis, there is probably no direct connection between the pigmentation and the fibrosis present. Mavrogordato[36] calls attention to the fact that the lungs in advanced silicosis from

hard rock mining are almost as black as those of coal miners. He thinks this is due to the fact that carbonaceous dust is prevented from being eliminated by the silicotic fibrosis, because the lymph circulation is interfered with, whereas, ordinarily if inhaled in the same quantities without the presence of silicotic fibrosis it would be largely eliminated. He intimates that dust cells containing coal and some other dusts die and do not form fibrous tissue. (See later discussion on coal miners.) Middleton[33] states that dark pigmentation is due to inert dusts like coal. Watt, Irvine, Johnson and Steuart[18] state that the South African mine air contains many pigmented particles, mainly carbonaceous, from smoke, coal and other sources, and that in areas of dust fibrosis in the lungs increased pigmentation is commonly found, but such dust is inert and plays no direct part in the production of the fibrosis. The pigment particles may play an indirect part in fibrosis by tending mechanically to block the lymph channels. At autopsies they found that the amount of pigmentation was not necessarily an index to the amount of fibrosis present.

ROENTGEN-RAY STUDIES

It is generally conceded that a properly conducted roentgen-ray examination is the most satisfactory and usually the most accurate means of studying the effects of dust upon the lungs of the living subject. A clinical study is also essential, especially in determining the capacity of the individual for work and the presence of a coincident tuberculosis. Most of the literature, even from the roentgenologic viewpoint, deals with pneumoconiosis as produced by the inhalation of silica. Roentgenologic studies suggest that probably other dusts than silica may produce fibrous changes in the lungs, so that the individual may be regarded as having pneumoconiosis. It is our purpose in connection with the roentgen-ray aspect of the condition to present our experiences with a large number of dusts. We have assumed that silica is not the sole etiological factor in pneumoconiosis, but in this we may be wrong. Such an assumption should be substantiated by appropriate dust analyses in order to establish a fact.

The reliance placed upon roentgen-ray studies is well expressed by Watkins-Pitchford,[38] chairman of the South African Miners' Phthisis Medical Bureau. He states that it is not safe to venture upon a positive diagnosis of silicosis without the assistance of "a technically satisfactory radiograph," which is so essential in all diagnostic roentgenograms of the lungs. He is authority for the very frequent confirmation of the accuracy of roentgen-ray findings by autopsies, which, fortunately are permitted by law in South Africa. Doubt exists in only the very earliest stages. It is a well-known fact that the first stage of pneumoconiosis may be simulated roentgenographically by several other conditions, and Watkins-Pitchford is correct in his statement that only when the specific appearances of the fine mottled stage are found is it invariably safe to venture a positive roentgen diagnosis, with the one exception of cases under observation for some time in whom the normal hilum and trunk shadows and spongework of interlobular septa are becoming more pronounced with repeated examinations. Steuart,[19] roentgenographer for the South African Bureau, regards the roentgen appearances as so striking that a glance at the roentgenogram is sufficient to visualize the exact changes in the lungs. At the time of writing he had filed over 200,000 roentgenograms of 57,000 individuals.

The technique of the roentgen-ray examination is of some importance. As we are studying fine details in the lungs, for the most part, the same care is advisable as in the examination of tuberculous lungs, and, in fact, tuberculosis may play a part in the appearances to be interpreted. We cannot too strongly urge the use of stereoscopy. Speed of exposure is a requisite in order to avoid all blurring due to heart and vessel pulsation. In a condition in which fibrosis plays the most important part, pulsation is especially likely to blur details. Many of the advanced silicotic individuals are extremely dyspneic and it is frequently very difficult for them to abstain from breathing, even for a second

or two. It is not advisable to ask such individuals to take the deepest possible inspiration, especially the extremely dyspneic ones, for the same reason.

The roentgenoscopic examination is often of great value, especially in determining respiratory capacity and observing the effects of fibrosis upon the diaphragm. In most of the advanced silicotics the diaphragms are nearly or quite immobile because of the inability of the fibrotic lung to expand or from pleural adhesions or both. Very frequently, in the less advanced cases, we are able to judge the extent of general fibrosis better from the effect upon the diaphragms, roentgenoscopically studied, than we can from the roentgenogram.

ROENTGENOLOGIC STAGES

It seems logical that the stages of progress of the fibrosis in pneumoconiosis should be based upon roentgen-ray appearances. A clinical classification would be based largely upon progressive dyspnea and capacity for work, and, as a matter of fact, these follow very closely the roentgenographic appearances.

In 1917, Lanza and Childs[3] presented the first American classification of stages of the condition based upon roentgen-ray findings. This has stood the test of time in so far as appearances are concerned, although there have been some changes in opinion as to the pathological features. They divided the progress of the condition into three stages, which may be briefly described as follows: *First stage:* The root shadows are denser and more extensive, and may show nodules; the trunk shadows are increased in density and breadth, with numerous punctate deposits of varying size along them; the appearance is symmetrical on both sides at first, but not so later; there is no difference in diaphragmatic excursion. *Second stage:* In addition to the foregoing there are found fairly symmetrical small, circumscribed, dense areas throughout both lungs, and later larger masses accumulate, usually at the lower part of the upper third, about the level of the root shadows; the domes of the diaphragms seem accentuated. *Third stage:* This differs from the second only in the extent of lung involvement, indicated by increased numbers of deposits and more massive grouping. They then go on to describe more advanced fibrotic changes, with heart, vessel and tracheal displacement and restricted diaphragmatic excursion which they were inclined to ascribe to a tuberculous complication, but which we now know may be due entirely to advanced silicosis, although tuberculosis may be a factor. This classification, with a few minor changes, answers the purpose admirably today.

During the same year, one of us, with Miller and Landis,[1] without any knowledge of the work of Lanza and Childs or others, based a similar classification, differing only in minor details, upon a study of the effects of dust inhalation in several industries. The appearances during the progress of the condition are so strikingly different that anyone is likely to select practically the same classification.

In 1916, Watt, Irvine, Johnson and Steuart[18] described three *pathological* stages as early silicosis, intermediate silicosis and advanced silicosis, with very similar roentgen-ray appearances in each as in the three just described. Mavrogordato[36] states that at the present time in South Africa silicosis is legally divided into three *clinical* stages: (1) *ante primary*, in which the earliest detectable physical signs are present, but there are no roentgenological changes; (2) *primary*, in which definite physical signs are present and capacity for work is impaired, though not seriously or permanently; and (3) *secondary*, in which capacity for work is seriously and permanently impaired. We know that the members of the South African Commission are keen diagnosticians and we cannot doubt the statement that physical signs are detectable before there is any roentgen-ray evidence. If this be true, as Mavrogordato states, that the roentgenogram will not detect the condition prior to fibrosis, whereas clinical signs will, possibly our failure to show prefibrotic evidence of the condition is due to the fact that there is no surrounding zone of congestion as is present in tuberculosis. Steuart,[19] roentgenologist for the South African Commission, states that

the legal clinical stages usually correspond to the roentgen-ray stages, but not always. He describes the following two roentgen-ray stages: *First stage:* The hilum and trunk shadows and linear marks are increased in prominence, but the appearance is not characteristic. *Second stage:* There is an apparent segmentation of linear markings which now present themselves as numerous small, discrete but clearly aggregated miliary nodules. The change from reticulation to nodulation usually progresses uniformly throughout the entire lung, and as the condition advances the nodules grow in size but apparently decrease in number. In addition, the grosser indication of the supervention of a tuberculous complication is the appearance of one or more areas of "clouding" in the hitherto uniformly mottled lung. These clouded areas are at first usually unilateral, and if in both lungs, they are usually not symmetrical. We have usually classed such appearances as third stage changes, and while in many instances they may be due to coincident tuberculosis we doubt very much that they always are. This phase of the condition will be considered more in detail later.

Nicholson[50] describes the following stages in England: A pre-roentgen-ray stage such as is recognized in South Africa, followed by first, second and third stages which are practically the same as those with which we are familiar. Purdy[41] states that the anteprimary stage of silicosis was legally recognized in South Africa in 1919. In the same communication he also states that the Broken Hill Commission of Australia laid down the rule that all miners should be systematically examined roentgenologically and if any abnormality was found, the findings demanded a physical examination. This would seem to give first importance to the roentgen-ray examination. Smith[60] describes the following roentgenologic stages in the Broken Hill district: (1) An increase in hilum shadows due to peribronchial thickening at the roots and to glandular enlargements. (2) A generalized nodular increase in the reticular shadow due to fibrosis surrounding the bronchi and bronchioles, and small areas of fibrosis whose blood and lymph supply is cut off by pressure of the increasing peribronchial fibrosis. This is the characteristic stage, and clinical signs are found only when roentgenographic appearances are well developed. In this statement he does not seem to share in the diagnostic opinion of the South African examiners. In his experience the roentgenogram has played the greatest part in diagnosis. Many autopsies checked their roentgen-ray findings.

Jarvis[11] reports a much more detailed and comprehensive classification among the granite workers at Barre, Vermont, as follows: *Stage A*: Granite pneumoconiosis, with increased prominence of hilum and trunk shadows; *Stage B:* Granite pneumoconiosis, with linear markings prominent at the periphery; *Stage C:* Granite pneumoconiosis, with a homogeneous haze over the lung between the linear markings; *Stage A:* Granite fibroid pneumoconiosis, with fibroid areas not larger than 3 cm.; *Stage B:* Granite fibroid pneumoconiosis, with fibroid areas not larger than 6 cm.; *Stage C:* Granite fibroid pneumoconiosis, with fibroid areas larger than 6 cm. He also gives an additional classification of tuberculous cases. While this classification is a working diagnostic and clinical basis for the peculiar conditions found among granite workers, and is a very excellent one, it would seem unnecessarily complicated for general use.

In later communications, Jarvis[12,13] presents his six stages in a somewhat different manner, based upon serial roentgenographic studies and conforming to intimate pathological processes: (1) Increase in hilum extent and prominence due to continued lymphatic drainage from the periphery hilumward. (2) Increased prominence of trunk shadows, due to hyperplasia resulting from the passage of the irritant through the lymphatics. (3) Appearance of small densities at the locations of the larger lymphoid deposits at the points of division of the bronchial tree as a result of their filtering function. (4) Appearance of fan-shaped areas at the lung periphery, indicating the reception of more dust than can be drained out of these areas hilumward. (5) A homogeneous

haze at the periphery, indicating a pleural lymphatic detour. (6) A gradually spreading cloud over the lung field toward the hilum. The author realizes that these stages are not always so progressively clean cut, but are likely to overlap.

For a satisfactory general classification we have seen no reason to change from the one suggested by Lanza and Childs, and we have followed theirs rather closely. A more detailed description of the three stages, together with the pathological processes responsible for each, follows:

FIRST STAGE

Roentgenographic Appearances. This stage is characterized by a very definite increase in the prominence and extent of the hilum shadows of the central zone, a thickening of the trunk shadows of the midzone and an increased prominence of the linear markings of the peripheral zone. In the hilum shadows will frequently be found evidence of many quite large tracheobronchial lymph nodes. The increased thickness of the trunk shadows is usually fairly uniform, although it is most noticeable at the bases and next to the apical regions. The outermost basal trunks are not so noticeably affected, as a rule, as the more mesial ones. This may be due, in part at least, to the fact that this portion of the lung, as stated by Miller, is drained, with the adjacent portion of the pleura and the diaphragm, through the ligamentum pulmonale to the preaortic lymph nodes, and not through the hilum. Lymphatic drainage may be less blocked for this reason.

Jarvis[11] divides the trunk shadows and linear markings into three groups of linear densities. Those of the first division, the broad ones in the hilum zone are most noticeable, of course, toward the bases. The second division, in the midzone, which is not the same as the midzone adopted by the Normal Chest Committee appointed by the National Tuberculosis Association,[62] he describes as having a geometric arrangement enclosing triangles in which fan densities may develop. The third division, the linear markings in the third or peripheral zone, he regards as the last to appear as a network of threads and forerunners of thickened pleura, which is so frequently found in the second and third stages of pneumoconiosis. He thinks these last densities are interlobular septal markings.

Interference with diaphragmatic excursion, which is a prominent feature of the second stage and still more of the third stage of the condition, is not noticeable in the first stage, except that frequently there is noted a restricton of the inner half of the diaphragm at full inspiration, as though this portion were held up in its descent by the portion of the lung containing the most prominent descending trunk shadows. It is probably the fibrosis along the bronchial tree represented by these thickened trunks that is responsible for a certain amount of the beginning inelasticity of the lung. This finding is an example of the importance of roentgenoscopic study in determining respiratory disability, which, should it occur early in the individual's occupation, should be a warning of what may be expected to follow.

These appearances of the first stage may possibly be produced by many inorganic dusts besides silica, or by organic dusts when inorganic material is mixed with them. Silica, however, will cause the changes in a comparatively short time, whereas other dusts may require several decades. Third stage changes are probably always due to silica.

The appearances of the first stage are by no means characteristic of pneumoconiosis alone. They may be simulated by passive congestion from cardiac decompensation, acute and chronic respiratory infections, chronic bronchial catarrh, the irritation from certain gases, and, to a less extent, by metastatic and rarely primary malignancy, usually carcinoma. The exact determination of early first stage changes is by no means as simple a problem as it may seem, first, for the reason just mentioned, and, secondly, because the hilum and trunk shadows in the adult are subject to so much variation within practically normal limits, depending upon age and environment, as in the city dweller. In childhood normal variations

are naturally not so marked and abnormal ones are easier of detection.

There should usually be no difficulty in detecting a coincident adult tuberculosis in this stage, provided it could be detected in the absence of pneumoconiosis in the same individual.

FIG. 1. Advanced third stage pneumoconiosis with extensive areas of massive fibrosis in both lungs. Male, aged fifty-eight. Had worked as a tool grinder for seven or eight years and afterwards as a farm laborer. Clinically the chief complaints and findings were abdominal, with, in addition, loss of weight, dyspnea, cough and profuse mucopurulent expectoration. The clinical history was lost and we have no knowledge of the sputum examination. The chest findings are essentially those of pneumoconiosis. We believe this case had an acquired eventration on the left side possibly due to phrenic nerve paralysis caused by the extensive fibrosis. This was suggested even more by the gastrointestinal examination than by the chest study. The rather heavy diaphragm arch excluded the likelihood of a life long eventration.

Pathology. The roentgen-ray appearances of this and all other stages are due almost solely to fibrosis or lymph block, and little if any to the dust itself, unless possibly when the dust is largely metallic. The first stage appearances correspond exactly to the early pathological changes. The preponderance of dust cells that have reached the tracheo-bronchial lymph nodes, resulting in enlargement and fibrosis of these glands causes the enlarged and denser hilum shadows. The direction of lymph flow being hilumward, actual fibrosis is

next mostly perivascular and peribronchial, due to dust cells lodging along the perivascular and peribronchial lymphatics, largely in the lymphoid nodules at first, and later outside of them. This produces the thickened trunk shadows.

The case as now observed may be one in which a large amount of silica has been inhaled for a comparatively short time, or a small amount of silica or other dust for a long period. Lymphatic obstruction may or may not be nearly complete. Interalveolar, interlobular, and possibly some subpleural fibrosis may have begun at this time, and may be noticeable as an increased prominence of the finer linear markings and a very slight pleural haze, observable only in very clear roentgenograms. Such appearances denote a transition into the second stage. Jarvis[11] describes these latter appearances as forerunners of thickened pleura.

SECOND STAGE

Roentgenographic Appearances. This stage is characterized by a typical mottling throughout the lungs due to small fibrotic nodules varying in size from a pinhead to a pea. The small shadows are more or less uniformly and rather symmetrically arranged except at first. In the early period of development of this stage we have almost invariably found the spots appearing first on the right side, around the root of the lung. The mottling becomes quite perceptible in the right lung before it appears on the left side (Fig. 2). The predominance is noticeable on the right side until the mottling becomes quite general throughout both lungs, and then there is usually no appreciable difference on the two sides. The earlier appearance and predominance on the right side is regarded as due to a larger amount of air going to the right lung. The mottling is always more marked around the roots and less at the apices and bases, especially the outer portions of the latter, probably for the same reason as was mentioned in connection with lessened intensification of trunk shadows in the same localities. The exact appearance or definition and the density of the small shadows depend, to a large extent at least, upon the quality and quantity of the inhaled dust. The densest and most sharply defined nodules we have found in metal grinders, in whom fibrosis may be very rapid. In occupations in which the fibrosis is comparatively slow, as in most coal miners and potters, the nodules are not quite so dense or sharply defined, and still less so in cement and asbestos workers.

Diaphragmatic excursion is frequently interfered with in this stage, although not to the same extent as is so frequently observed in the third stage. When the fibrotic nodules, indicated by the small shadows,have progressed to a considerable size,and it is a question as to whether the case should be classed as second or third stage, we have sometimes reached a decision by observation of the diaphragms. In the second stage, the descent of the diaphragm may be restricted only in the inner half, as has occasionally been noted even in the first stage, but there may be a definite restriction throughout its entire extent. When irregularities in contour or sharp peaks at the ends of the trunk shadows occur the decision is usually in favor of the third stage.

Theoretically one would expect the nodular fibrosis of the second stage to be superadded to the first stage appearances, but this is not always the case. In many instances the hilum and trunk shadows show little or no increased prominence in second stage cases. At first we were inclined to ascribe this to an obscuring effect of emphysema, which we feel certain is indicated in roentgenograms of cases of this stage, but this does not seem to be a sufficient or the sole explanation. Watt, Irvine, Johnson and Steuart[18] state that emphysema may obscure the typical appearance. A more plausible explanation, though possibly still not the correct one, would seem to be somewhat along the following lines: As the more peripheral lymphatic channels become blocked by fibrosis, lymph drainage toward the hilum becomes largely discontinued, and few dust cells reach the central portions or root regions. As previously stated, it has been proven that a lung can clear itself of dust and dust cells to a certain extent. This may occur in the central portions,

with some clearing up of the hilum and adjacent trunks. We would not expect fully formed fibrous tissue to disappear, however. The clearing up process would seem most likely to occur in cases in which the shadows were due mostly to dust cell collections and lymph stasis, and in the more rapidly developing cases, therefore. This really seems to be the type of cases which show evidences of clearing. Jarvis[11] calls attention to the fact that as long as the lung fields remain clear the hilum maintains its first stage individuality, but when the appearance of thickened pleura overspreads the peripheral zones, the hilum shadows disappear to a large extent. He thinks there must be some connection between the two, and that possibly the hilum shadow in these more rapidly progressing cases is largely lymph stasis which disappears when the lymph block indicated by pleural thickening, is

FIG. 2. Moderately advanced and typical second stage pneumoconiosis in a hard coal miner of thirty years' occupation. Exact duties not known. Male, aged forty-eight. Clinical data unimportant. Fair diaphragmatic excursion. Rather coarse "soft" spots of nodular fibrosis fairly evenly distributed throughout both lungs, but still a little more extensive on the right side. The first stage appearances are not prominent. Note the freedom of the left base from nodules.

established. This may be true, but it seems just as plausible to believe that the large intensified hilum shadow is due to cell hyperplasia and fibrosis, and we know, first, that there is a time interval between the lodgment of dust cells and their development into fibrous tissue, and, secondly, that dust cells can disappear. The thickened trunks can be more reasonably accounted for by lymph stasis than the enlarged hilum.

The appearances of the second stage of pneumoconiosis are so characteristic that the condition at this period is not likely to be confused with any other by one who is familiar with it, except possibly a miliary or bronchopneumonic tuberculosis, or in some instances, a case of the puerile type. The characteristic features of the second stage as to its general distribution throughout the lungs, the similarity of the small shadows as to shape, size and density, their greater numbers around the roots and lower portions instead of the apices, together with the clinical history, absence of fever and other constitutional evidences of tuberculosis and possibly the sputum examination should serve to render the diagnosis comparatively easy.

The black pigmentation of the sectioned silicotic lung is apt to be associated, by one not entirely familiar with the pathology of the condition, with the nodular appearance of the roentgenogram. While the pigment is in the areas of fibrosis, it has little or nothing to do with the roentgenographic shadows, which are due to the fibrosis or stasis, except possibly in the case of metallic dust. The pigmentation, so conspicuous in the coal miner or fireman, is a coincident associate of the fibrosis producing element of the dust.

The differentiation of a *superimposed* tuberculosis in this stage is not always so easily made as one is frequently led to believe. In an early period of the second stage an early apical tuberculous lesion may be readily differentiated in most instances, but later on, particularly as the condition approaches the third stage, the diagnosis becomes more difficult. Of course the extreme apices usually remain comparatively free of silicotic changes until late, which helps materially in the diagnosis of a tuberculous process in the apices.

Pathology. It is evident that the fibrotic nodules characteristic of this stage have their incipiency in the lymphoid deposits found along the lymphatic channels and so fully described by Miller.[27–32] So far we have been dealing with roentgenographic appearances due to the passage of dust cells hilumward and the fibrosis which results there. All of this time, however, some of the cells have found lodgment in the small lymphoid deposits found at the junctions of the peribronchial and perivascular lymphatics and more peripherally. As these deposits accumulate dust cells they grow larger, and fibrosis subsequently takes place. Some develop into small nodular fibrotic areas faster than others. When they become large enough of themselves, or with the addition of the surrounding areas of lymph stasis, to become visible they will cast definite shadows on the roentgenogram. In some films they appear as innumerable very small shadows, while more frequently there are a lesser number of somewhat larger ones.

The sharply defined spots of the rapidly progressing silicosis of the hard rock miner, granite cutter and others are probably due to a rather sharp limitation of the fibrosis to the area of the lymphoid deposit, whereas the "soft" or "fuzzy" spots of the slowly progressing process of the coal miner, potter, cement and asbestos worker may be the result of a surrounding interstitial fibrosis permitted by the longer time factor. This is purely conjectural, of course.

After a time many of the small nodules no doubt coalesce to form larger ones. As long as the appearance remains that of discrete nodulation, pinhead to pea size, without diffuse haze or localized hazy areas among the nodules or marked diaphragmatic restriction or deformity not due to other causes, the case may be regarded as belonging to the second stage.

THIRD STAGE

Roentgenographic Appearances. This stage is characterized by a diffuse fibrosis, of which there are three fairly definite

and distinct appearances, although all of them may be observed in the same case. (1) The larger nodules, where most prevalent, may coalesce into very much larger and irregular masses or be found close together with more or less haze between (Fig. 3). In this type of case it is sometimes difficult to draw the line between the second and third stages. The appearance of the diaphragm may help. (2) A more frequent appearance is that of a more or less diffuse fibrosis with evidence of rather definite nodules still present (Fig. 4). This appearance does not differ very greatly from the fibrotic process which represents the end result of an extensive bilateral pulmonary tuberculosis (Fig. 5). Although we may be able to state definitely that such a case is one of pneumoconiosis from the history and

FIG. 3. Definite third stage pneumoconiosis in a hard coal miner of thirty years' occupation. Exact duties not known. Male, aged fifty. Important clinical data bearing on chest condition—cough for five months, with black sputum, dyspnea, emphysema, râles at bases. Fair diaphragmatic excursion, although the left diaphragm shows well-marked sharp peaks. The diffuse nodular fibrosis of the second stage is still evident as large dense spots. In some areas these have become conglomerate, and in others there is more or less haze between the nodules. The prominent trunk and hilum shadows are still in evidence.

persistence of nodules, no one can be certain from the roentgenogram alone whether or not a superimposed tuberculosis is present.

We have observed this but once. It is often difficult to distinguish between a pleural thickening and a lung density from fibrosis or lymph block. In post-

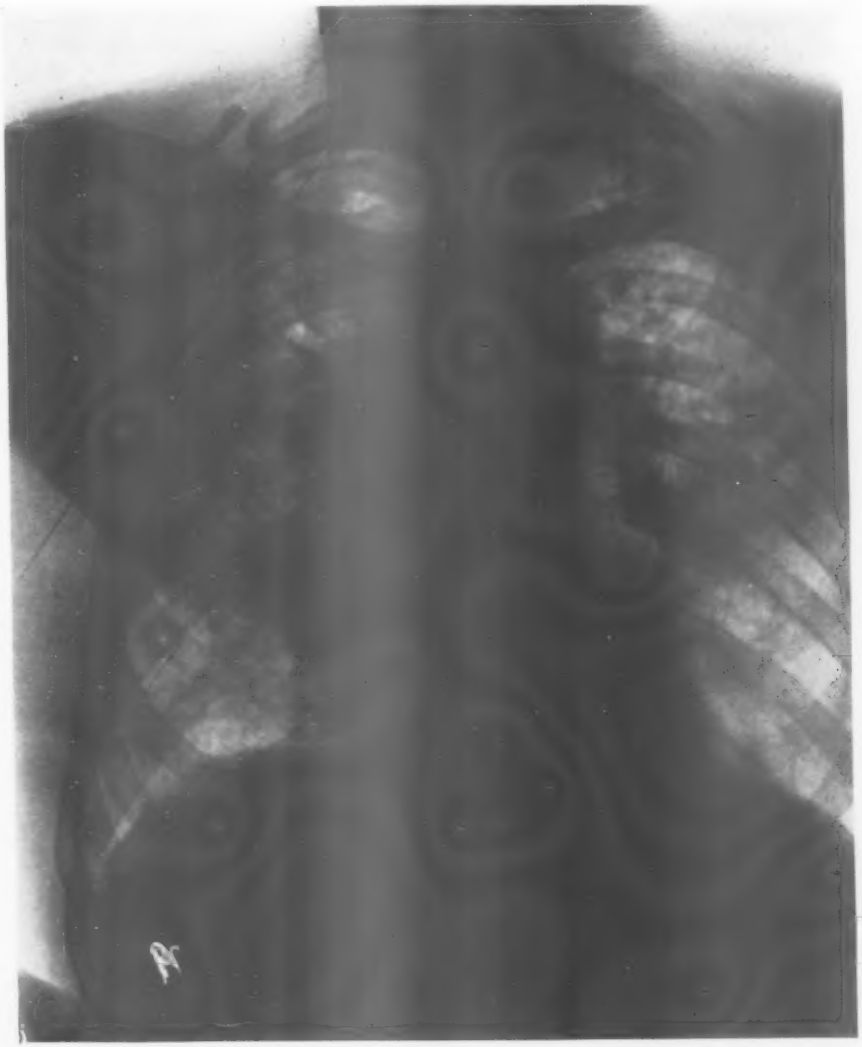

FIG. 4. Well-advanced third stage pneumoconiosis in a flint grinder in a sandpaper works, ten years' occupation. A very rapid process, even though he wore a mask, which would not, of course, keep out the dangerous minute particles. Male, aged forty-one. Important clinical data—cough for three years following influenza and has lost 50 pounds in weight. In a tuberculosis sanitorium three months, but repeated sputum examinations negative. Impairment to percussion right upper lobe and upper portion right lower lobe. Fair diaphragmatic excursion, as would be expected from the appearance. The right lung is most affected and shows a rather diffuse fibrosis with some evidence of nodular fibrosis still remaining. The left lung is similarly affected to a less extent. Tuberculosis is not excluded roentgenologically, but pneumoconiosis is the essential condition. Pleural thickening is probably present.

In both of these types evidence of pleural thickening may be found, especially peripherally, in the axillary regions. Jarvis has described pleural effusions. pneumonic or simple pleurisies followed by thickening, the latter is usually found at the base and is quite characteristic, especially when it produces a diaphrag-

matic adhesion. The thickening accompanying pneumoconiosis is usually elsewhere, higher up, and does not present the same appearance.

(3) The third appearance, and one very frequently encountered in this stage, is a very striking one, and most interesting from the pathological as well as the roentgenological standpoint. It is that of *massive fibrosis*, with the appearance of extensive pulmonary consolidation. There is usually one such area in each lung, rather symmetrically located (Fig. 6); occasionally more than one (Fig. 1); sometimes it may be unilateral (Fig. 7); or more often the area is more extensive on one side than on the other (Fig. 8). These areas are apt to be subapical in contradistinction to the consolidations of tuberculosis (Figs. 9 and 10). Stereoscopically the fibrotic areas are found to be well within the lungs, although they are likely to extend to the pleural surfaces. Probably they are covered by thickened pleura, although this cannot be proven roentgenographically. Watt, Irvine, Johnson and Steuart[18] found the overlying pleura usually thickened and adherent at autopsy.

Associated with massive fibrosis dense fibrous bands can usually be seen extending in various directions, but usually downward (Fig. 6), and some of them are quite wide and connected with marked deformities of the diaphragms (Figs. 1, 7, 12 and 16). The bands passing downward have the appearance of thickened trunks. In the majority of third stage cases the trunks are inconspicuous, especially in

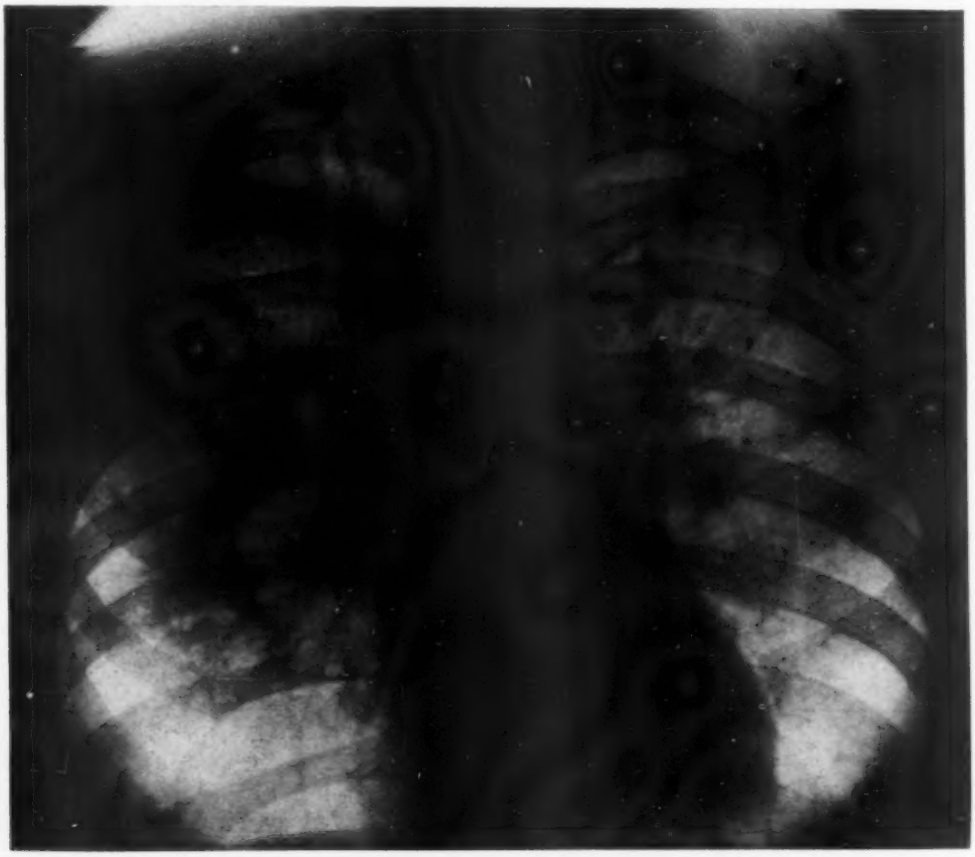

FIG. 5. Chronic fibroid tuberculosis with very little activity. Male, aged forty-four. The right lung is mainly involved and contains a large, irregular cavity supposed to be bronchiectatic. The appearance does not differ so materially from that of Figure 4, and a clinical record of both cases is essential before attempting to render an interpretation.

the first two types. The diaphragmatic deformities vary from slight inequality in contour on deep inspiration to sharp peaks which may be scarcely noticeable to extremely deforming. The sharp peaks appear to be at the points of attachment of fibrous bands or trunks. Under such circumstances the diaphragm is definitely restricted, and frequently does not move at all when viewed roentgenoscopically, and the ribs very little. Such individuals are extremely dyspneic, and satisfactory examinations require very rapid exposures. Emphysema, especially of the bases, is almost always present, and usually the roentgenograms are hard to reproduce because of resulting extreme contrast.

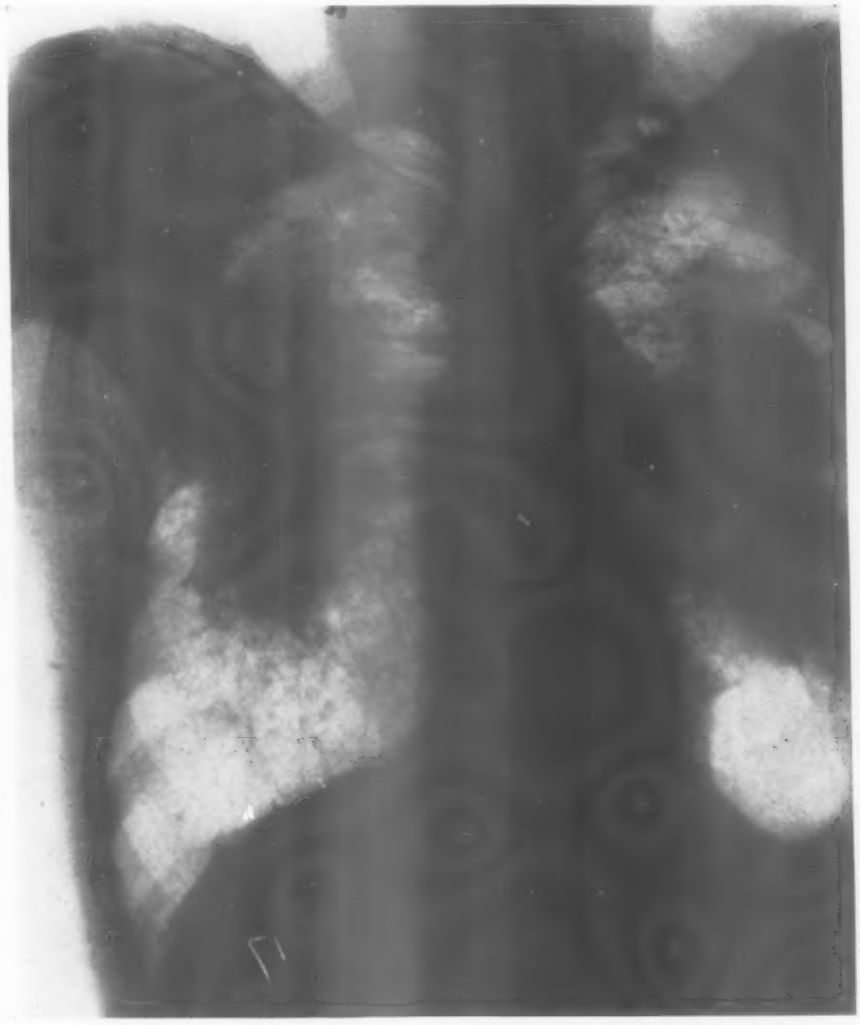

FIG. 6. Third stage pneumoconiosis with massive fibrosis in a coal miner of nineteen years' occupation. Male, aged forty-three. Exact occupation not known, but we would pick him out as a driller. The appearance resembles that of consolidation, rather symmetrically placed in the subapical area in each lung. This is a very common occurrence. Writers with considerable autopsy experience regard such areas as due to tuberculo-silicotic fibrosis, although the organisms may never be found in the sputum. Note the sharp peaks in the right diaphragm and the decided hump in the left and the distortion of the aortic arch. The bases are emphysematous, and the outer portions comparatively clear of fibrosis. No evidence remains of second stage nodules or of first stage hilum prominence.

In the worst third stage cases, especially those with massive fibrosis, the heart and great vessels may be considerably drawn

out of place, though not to the extent seen in some organized pleurisies.

Rarely any other conditions than tuberculosis produce fibrosis so extensive as is found in the third stage of pneumoconiosis (Fig. 11). The difficulties in the differential diagnosis between tuberculosis and the third stage, and in the detection of a superimposed tuberculosis have already been referred to. The third type of this stage requires special mention in connection with tuberculosis and will be discussed later under pathology in the paragraphs which follow.

FIG. 7. Third stage pneumoconiosis in a quartz miner of nine years' occupation, but a motorman for the past ten years. Male, aged forty. Important clinical data—recent cough, expectoration, slight temperature and loss of 6 pounds in weight. Pulmonary tuberculosis suspected, but cannot be proved or disproved from appearance, in view of the occupational history. Note the peaks in each diaphragm, due to the fibrosis. Both were very much restricted in movement. The entire appearance might be due to tuberculosis or to silicosis. Unilateral massive fibrosis is frequently seen, as in the right upper lobe, in silicosis, and if due to this cause would very likely be of tuberculo-silicotic origin.

Pathology. With the exception of the massive fibrosis stage it seems likely that the diffuse fibrosis results from either a conglomeration of the small nodular fibro-

sis of the second stage or an advance of the interstitial fibrosis mentioned under pathology, or both together, with one or the other in the ascendancy. There seems to be some difference of opinion as to the exact pathological procedures leading up to the formation of the areas of massive fibrosis. The consensus of opinion seems to indicate that these areas are the result of a combination of silicosis and tuberculosis in many if not most instances. For a long time we regarded the areas of massive fibrosis as nontuberculous for the reason that in a number of cases presenting the appearance careful and repeated search for the tubercle bacillus in sputum failed to reveal any. It was not until we became familiar with the literature emanating from countries where autopsies were the rule on cases dying from advanced pneumo-

FIG. 8. Third stage pneumoconiosis in a potter of thirty years' occupation. Male, aged forty-seven. The chief complaint was bronchitis of six years' standing. Pneumoconiosis suspected. There is a large area of massive fibrosis on the right side and a much smaller one on the left. Very little evidence of second stage nodular fibrosis remains and the first stage hilum prominence has disappeared. The right diaphragm shows peaks and was very much restricted, but the left showed fair excursion. The outer portions of the bases are fairly clear.

coniosis that we became convinced that massive fibrotic areas were partly tuberculous in origin.

Watt, Irvine, Johnson and Steuart[18] state that a silicotic process seems intensified around a tuberculous area. They described these areas as arising homogeneously and not from a conglomeration of nodules, although they acknowledged that their origin from extensive coalescence of nodules or diffuse fine interstitial fibrosis was possible. They also

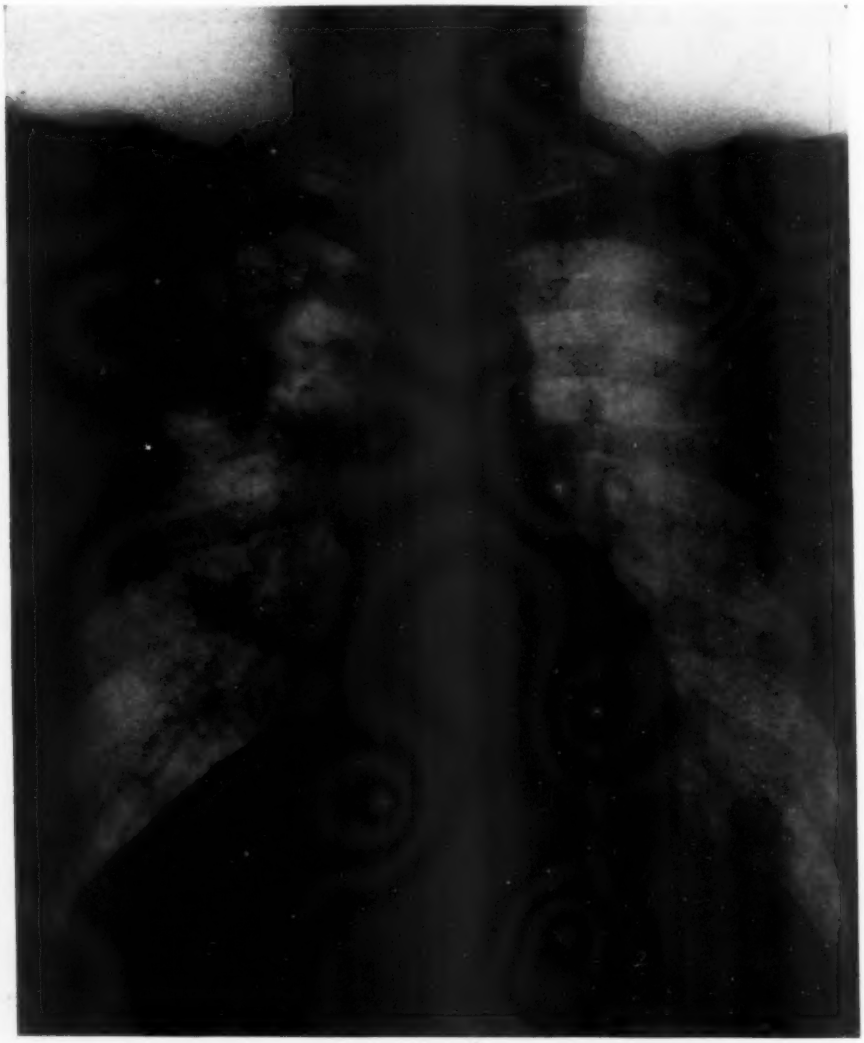

FIG. 9. Probably a second stage pneumoconiosis in a granite cutter of twenty-five years' occupation. Male, aged forty-five. The silicosis is not so far advanced as is usually seen in the granite cutter who uses the pneumatic tool for this length of time. The diagnosis of second stage is based upon a moderate nodular fibrosis seen in the films. The right apical lesion is probably tuberculous, as it contains a large cavity and the sputum was positive. Evidences of tuberculosis predominate in this case, and the right apical consolidation is out of proportion to any other evidences of advanced silicosis. First stage hilum and trunk shadow prominence are still very evident. There was good diaphragmatic excursion.

believed that a combination of the two processes was the common mode of origin of the areas of massive fibroid consolidation. stated that a chronic nontuberculous pneumonia might also be a helping factor. These views were based largely upon

autopsy findings. They found that caseation in these areas was uncommon, but did occur, but not when fibrosis was fully developed. Where caseation occurred they did find the tubercle bacillus. No caseation was found in hilum glands or nodules

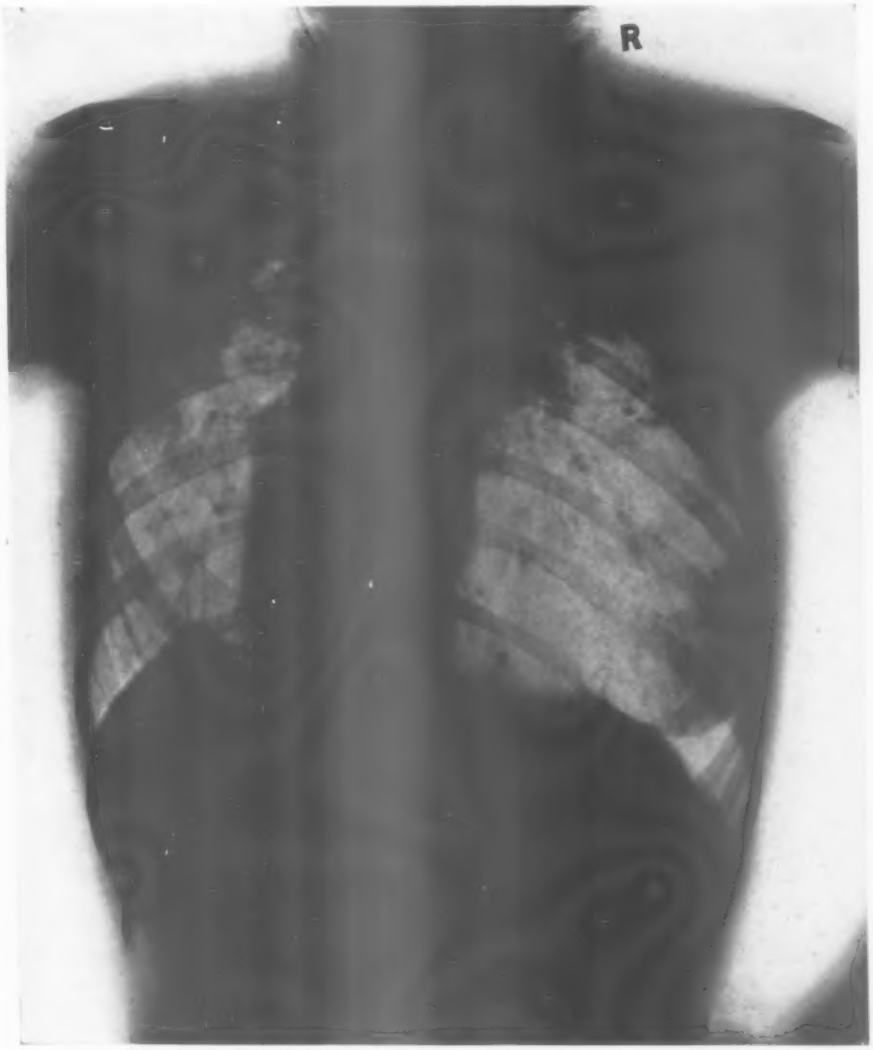

FIG. 10. Extensive fibrosis resulting from pulmonary tuberculosis. This is a case of chronic fibroid phthisis. Female, aged twenty-five. Note that the consolidations or densely fibrosed areas are apical and higher than usually found in silicosis. There is an absence of any resemblance to nodular fibrosis. Both diaphragms show peaks to which fibrous bands extend, and both were restricted. The mediastinal shadows are distorted.

in less advanced stages of pneumoconiosis. Dense massive fibrotic areas would occasionally break down, presumably because of cut off blood supply, and in such cases bacilli were sometimes found. Massive fibrotic areas occurring comparatively early in silicosis were regarded as certain evidences of tuberculous invasion. The density of fibrosis was regarded as a retarding factor against the spread of the infection. Hoffman[44] states that in experience with granite workers blocked lymphatics cut off nutrition and any infection may cause a fibrous area to break down. Cavities were frequently observed in granite cutters.

Mavrogordato[36] agrees with Watkins-Pitchford[38] and others[18] that the massive fibrotic areas are frequently an evidence of superimposed tuberculosis or other infection, and states as his experience that they do not occur without some infective agent, tuberculosis being a conspicuous but not the sole one. He describes the process, in brief, as follows: The massive fibrotic areas are formed primarily of nodules with the addition of the round cell infiltration and granulation tissue of the infective agent between them. In this internodular area fibrosis subsequently occurs as a result of the infective process rather than by the confluence of the small nodules of silicosis. His impression of roentgenographic appearances is that a fibrotic nodule is surrounded by apparently normal lung, but in case of infection it will be seen to be surrounded by a fringe due to lymphocytic and polymorphonuclear infiltration.

Gardner[23] in animal experimentation found that the simultaneous infection with mild strains of tubercle bacilli in the presence of granite dust silicosis caused much greater fibrosis, and that both together set up a reaction that neither alone could cause, each apparently reinforcing the other. The result was a chronic lesion that was slow in healing. The exact mechanism of the reinforcement was uncertain.

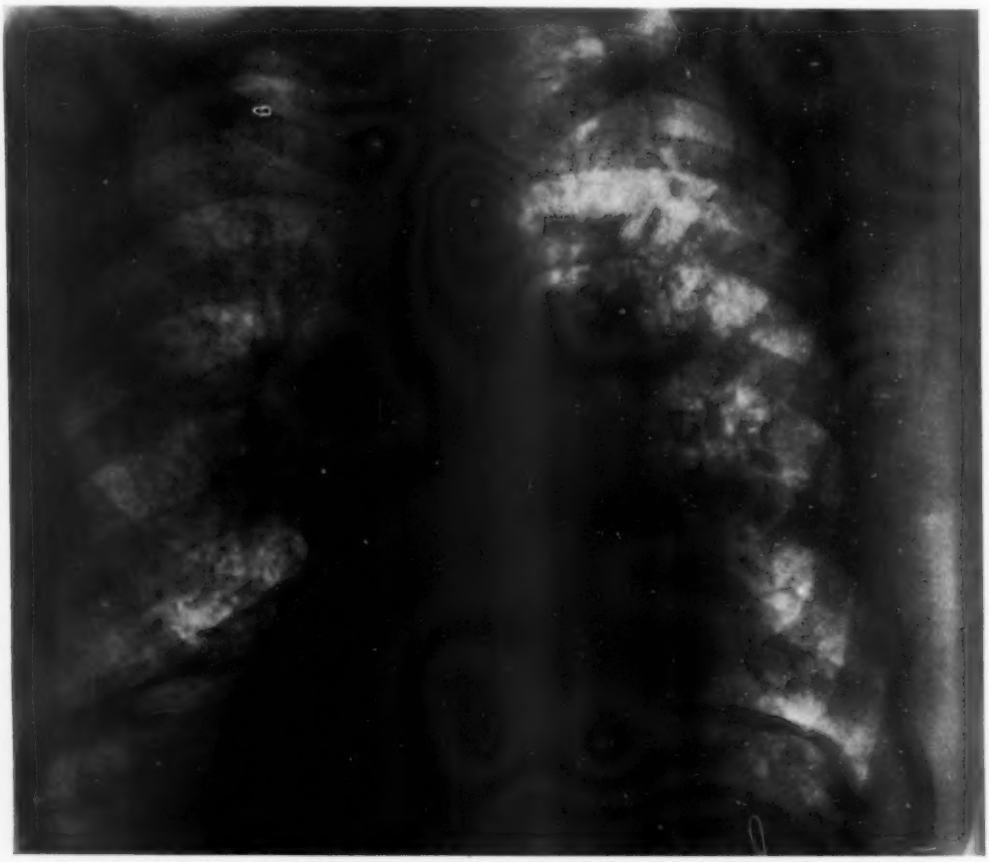

FIG. 11. Extensive fibrosis of both lungs due to a chronic process resulting from the inhalation of a vegetable burr during childhood. Male, aged twenty-nine. This was not a tuberculous case, based upon prolonged clinical study. An unusual feature was the fact that basal bronchiectasis could be readily detected roentgenologically. One would hardly be likely to confuse this case with silicosis.

COINCIDENT TUBERCULOSIS

This subject has of necessity been frequently discussed in connection with

various phases of pneumoconiosis. Certainly the two conditions are closely allied for the reason that pneumoconiosis undoubtedly may produce a fertile field in the lung for the tubercle bacillus, aside from any external and constitutional predisposing influences, such as hygienic surroundings. Tuberculosis is, therefore, one of the most important phases of the subject. The disabling effects of silicosis and the frequency of tuberculous infection and the resulting high mortality of the past have been the outstanding serious features of pneumoconiosis. At one time pneumoconiosis in its advanced stages was regarded as tuberculosis. More recently, it was supposed by some of experience limited to certain dusty industries that the condition protected the individual in a measure against the spread of tuberculous infection. While this may be true to a certain extent, as in coal miners, we know that it is not the rule. Tuberculosis is most likely to seriously affect those in whom silicosis is rapidly developing, and it can be controlled to a great extent, therefore, by appropriate measures directed against the rapid course of the predisposing factor.

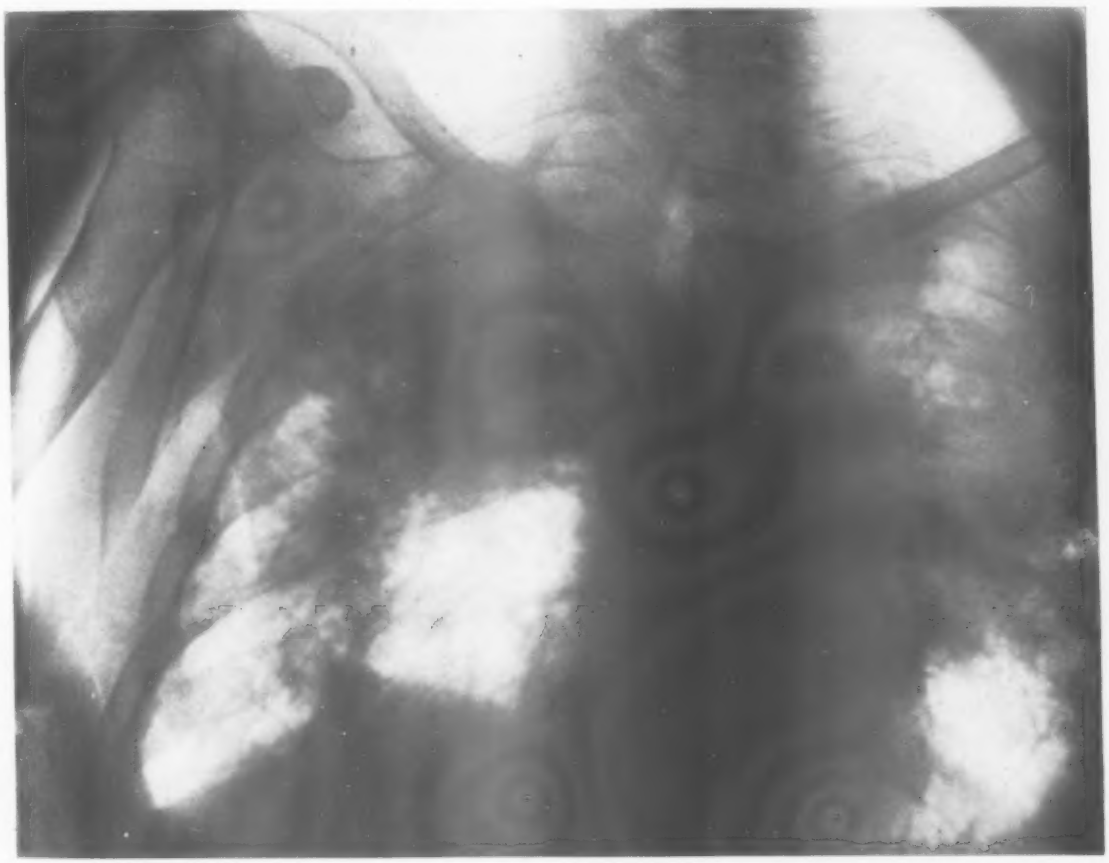

FIG. 12. Advanced third stage pneumoconiosis in a coal miner of thirty years' occupation inside the mines. Male, aged fifty-one. There are extensive areas of massive fibrosis. Although repeated sputum examinations were negative, these areas would probably be generally regarded as tuberculo-silicotic in origin. This man was intensely dyspneic and could scarcely stand. Both diaphragms were practically fixed. This is one of the worst cases we have ever observed.

Watt, Irvine, Johnson and Steuart[18] state that the liability to tuberculosis seemed to increase in proportion to the amount of silicosis present. In the earlier silicotic cases the more rapid tuberculous processes were more common, as tuberculous pneumonia, acute tuberculous broncho-

pneumonia or acute miliary tuberculosis. These were less commonly encountered and the more chronic manifestations of the disease were observed in men with advanced silicotic lungs. Tuberculosilicotic cases did not seem so apt to infect other individuals as cases of pure tuberculosis.

Bulletin 293, Bureau of Labor Statistics[9] states that tuberculosis apparently supervenes on an extensive silicosis among granite cutters usually after ten to fifteen years' exposure, and it is concluded that granite and all quartz dust predisposes to tuberculosis. Jarvis[11] states that statistical reports for twenty years up to 1919 conceded 86 per cent of deaths among granite cutters were due to tuberculosis.

Nicholson[50] speaks of a high tuberculosis mortality from silica dust in the Derbyshire district among workers in quarries, miners, and stone cutters working among rock, sandstone and other materials containing 85 to 96.4 per cent of silica. These workers frequently showed marked lung changes from silicosis in five to ten years. Middleton,[33] from a large experience with silicosis from various occupations, states that all evidences point to tuberculosis as a frequent cause of death among silicotics. Scurfield[56] in 1908 reported a high death rate from tuberculosis among grinders and cutters and others in Sheffield, ranging from three to six times the average death rate from the disease. It was higher in grinders. Ickert[59] studied 400 cases of pneumoconiosis with tuberculosis among the workers in copper and slate mines in the Mansfield district, Germany. All showed marked lung fibrosis with tuberculosis and positive sputum. They lived longer as a rule than others with tuberculosis alone, and usually died between the ages of fifty and sixty. The infection in their families was more active and they seemed to spread the disease.

Kerr[61] states that in 1906 Simmons investigated the Bendigo miners in Australia and found six times as many deaths from tuberculosis among them as in other individuals. Smith[60] has made some very interesting statistical studies including 8,966 Broken Hill (Australia) miners. He found that those who showed silicotic changes were twenty-three times as susceptible to tuberculosis as those who did not. Of 174 silicotic men without evidences of tuberculosis who were sent away from the mines by the Broken Hill Commission and subsequently watched, 37 developed tuberculosis in two and one-half years. The subsequent infection occurred mostly in those who settled in congested towns and cities. In all these cases the tuberculosis was an entirely new infection and not an activated old one.

Watt, Irvine, Johnson and Steuart[18] call attention to the fact that sputum is not so apt to become dried out in mines, and remains a menace for a longer time, so that miners are more exposed to infection. In South Africa, Australia and many other localities all tuberculosis suspects are excluded from mines and all other intimate contact with miners.

Mavrogordato[36] thinks that miner's phthisis has changed in type and roentgenographic appearances in recent years probably because tuberculosis has become a more conspicuous factor. It often kills the individual before he has had an opportunity to develop the old-fashioned silicotic lung because of the partial control of dust in the mines. Life is not shortened, but tuberculosis complicates now at a comparatively earlier stage of silicosis.

There are very numerous other writers on this particular phase of the subject, but space forbids quoting other articles than those which have been used for purposes pertaining directly or indirectly to the roentgen-ray aspect of pneumoconiosis. It has been, likewise, an impossible task to review all the literature covering all industries in which pneumoconiosis has been studied. It was our original intention in this communication to present the results of our own experiences in the roentgenological study of several hundred cases of the condition selected from several different industries. While the statistics we wish to present represent a relatively small number of cases compared to the enormous numbers studied in other countries, the group is rather large for an individual contribution originating in the United States.

COAL MINERS

Probably the largest general group of pneumoconiosis cases that is likely to be collected by anyone in the eastern portion of the United States, except where routine health investigations are being made, as by Jarvis at Barre, Vermont, is that of the coal miners. Our experience with the workers in this industry, aside from consulting work with the United States Bureau of Mines, has never been one of investigation, but only the examination of the occasional case which drifts into the larger medical centers. It has been limited, with the one exception just mentioned, almost entirely to the anthracite miners of Eastern Pennsylvania. We have examined 84 cases of definite pneumoconiosis among these miners. Six showed only first stage changes, 54 second stage, and 24 third stage appearances. Hard coal mining may, in some respects, be regarded as a somewhat hazardous occupation from the standpoint of pneumoconiosis, but not nearly to the extent of many other mining industries. Of all the workers concerned, only those in certain definite occupations are exposed to sufficient quantities of harmful dust to be regarded as being in any danger.

Of our cases, the earliest evidence of first stage changes was after three years' occupation, and the longest time at work and remaining in this stage was twenty years. The earliest second stage changes were found after two years' occupation, but the average was about six to eight years when examined. The longest occupation among second stage cases were two men at forty-two and forty-eight years, while twenty to twenty-five years seemed to be the average time of the older cases when examined. The earliest third stage case was ten years' and the oldest fifty-three years' occupation, and the average when examined was between thirty and forty years. Of course, these statistics mean little for the reason that we have records of only positive cases, and many negative miners were examined for one reason or another in whom no pneumoconiosis was found. Moreover, it is very important to regard the exact occupation of the individual in coal miners' statistics. The very dusty breakers are not nearly so harmful as the occupations of driller or digger. One man who worked in a breaker for eight years showed no changes whatever.

The statistics of the positive coal miners we have examined do not differ so much from those of Jarvis' granite cutters,[9] except a slightly longer time being required to produce like changes. But it must be borne in mind that only a small number of hard coal miners are exposed to dangerous dust as compared to a relatively larger number of granite workers. Anthracite miners' chests with advanced silicosis present much the same appearances as are found in hard rock miners, although a longer period of time is required to produce them. One of the worst cases of pneumoconiosis we have ever examined was in an anthracite miner of thirty years' occupation (Figs. 12 and 13).

Probably most of the reports on coal mine dust pneumoconiosis, especially outside of the United States, refer to soft coal mining, which is quite a different proposition from anthracite mining. We have interpreted chest roentgenograms of nearly 500 soft coal miners for the United States Bureau of Mines, and among these were comparatively few showing any very advanced pneumoconiosis. Exact occupation is again an important factor, and comparatively few soft coal miners seem to be exposed to dust of a dangerous character or quantity. The character of the mine as to rock and sand encountered is also to be considered. Almost invariably the driller or his helper were the ones to show the more advanced stages of the condition. In these men second stage appearances were frequently noted after six to eight years' occupation. By knowing the length of time at work we could usually pick out the roentgenograms of men in these particular occupations.

In addition to coal miners, pneumoconiosis may be found also among firemen or stokers. We have found fairly well advanced second stage changes in locomotive and marine firemen. One of the latter showed well advanced second stage changes after thirteen years' occupation. These men worked with soft coal and

presumably would not be exposed to the same amount of silica as the miner.

A very interesting feature of pneumoconiosis in coal miners is the apparent protection directly or indirectly afforded by the coal dust against tuberculosis. While many coal miners with pneumoconiosis do contract tuberculosis, the disease does not seem to be so prevalent among them as among many other classes of miners. There is a prevailing opinion among many physicians in the coal regions, and even with the laity to a certain extent, that the coal miner is more or less protected against tuberculosis. This disease is, of course, more apt to prevail among those who develop silicosis rapidly, but there seems to be less tendency to it among coal miners than in industries in which the silicotic development period is comparable.

Mavrogordato,[36] realizing these facts, has attempted to explain them. He believes that dust cells containing coal and clay die readily and are digested or undergo autolysis, leaving their dust extracellular *in situ*. By animal experimentation he was able to prove that simultaneous exposure to coal and silica dusts appeared to create a condition preventing fixation of the silica. If silica exposure preceded the coal dust this did not occur. He thinks that in coal miners the coal dust aids the lungs in dealing with the comparatively small amounts of silica present with coal, and the latter aids in silica elimination.

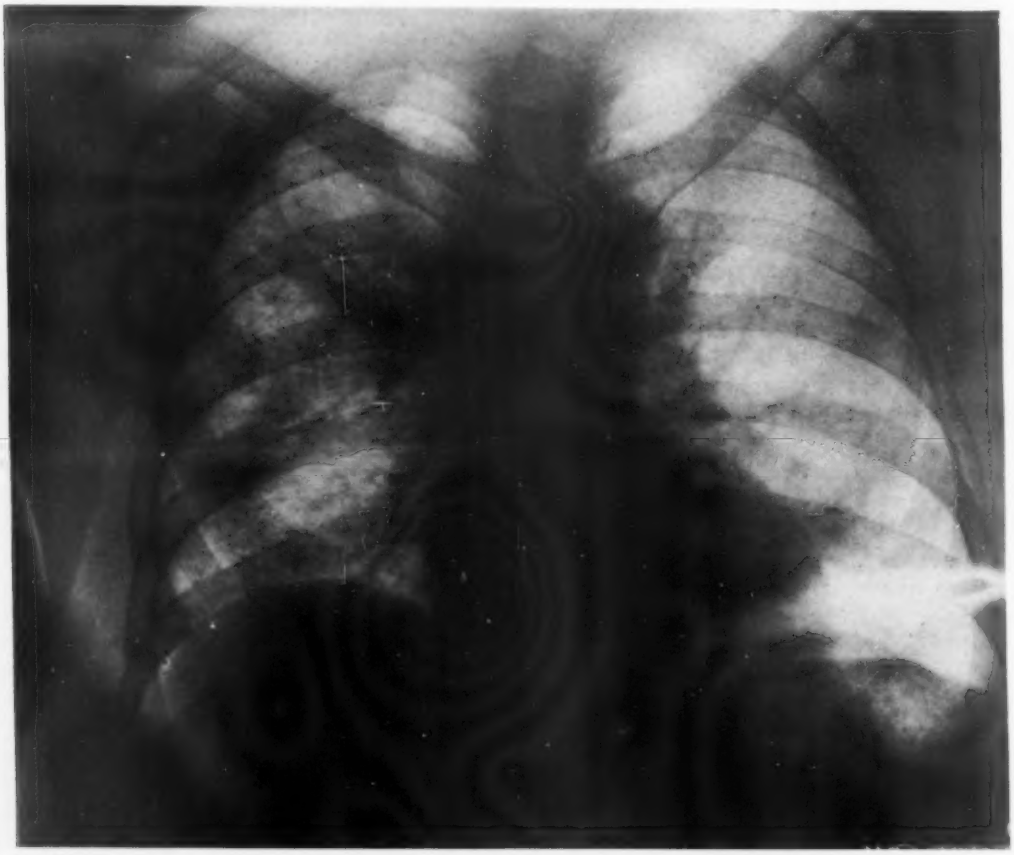

FIG. 13. Second stage pneumoconiosis in a coal miner of thirty-eight years' occupation outside the mines. Male, aged forty-seven. Compare with Figure 12. The predominating features in this case are almost as much of the first stage as of the second. Lymph block has not cut off drainage to the hilum region and the hilum shadows are very prominent. The second stage appearance is a diffuse nodular fibrosis throughout each lung indicated by very soft spots. There were no unusual chest symptoms in this case.

He states that coal miners in South Africa do not develop miners' phthisis (pneumoconiosis) any more, as a result of preventive measures.

Nicholson[50] speaks of the low death rate from tuberculosis among coal miners in the Derbyshire district and states that around Sheffield who die at an average age of thirty-nine or forty years, tuberculosis accounts for 75 to 84 per cent of the deaths. Purdy[41] after his review of 103 contributions to the subject states that it seems to be the consensus of opinion that some dusts like coal not only have no

FIG. 14. Third stage pneumoconiosis in a granite cutter of twenty-five years' occupation. Male, aged forty-six. The original roentgenogram indicates a diffuse haze from fibrosis through the right lung and evidences of thickened pleura. At the base conglomerate nodular fibrosis is becoming a massive fibrosis. The same appearance to a less extent is noted at the left base, where definite nodules can still be seen. It is unfortunate that nodules are so small as to be frequently lost in the reduction for illustrations. The fan-shaped areas described by Jarvis are to be seen along the left axillary border.

this is peculiar to coal mining all over the world. He regards coal as capable of exerting some protective action against tuberculosis. Middleton,[33] on the other hand, quotes Collis[40] as stating that with the coal workers working with ganister power of producing pneumoconiosis, but may even possess some inhibitory influence over phthisis. Myers[57] in studying the vital lung capacity among bituminous coal miners found no appreciable decrease in capacity below the age of 46 years, or

24 years' service in the mines. Any lower averages after these periods were attributed more to age than to occupation.

HARD ROCK MINERS

This group of workers is the most important in any of the dusty industries the world over because of the dangerous character of the dust and the number of individuals engaged. Our personal experience in examining such individuals is limited to one case (Fig. 7), although we have interpreted roentgenograms of about 600 miners for the Bureau of Mines and others. These included workers in gold, iron, zinc, lead, copper, cobalt and nickel mines. The appearances found have borne out the statements previously made in connection with such workers. A great amount of writing has been devoted to hard rock miners, and it may be summed up in the brief statement that where the rock contains a large percentage of silica, unless adequate protective measures are adopted, the miners, particularly drillers and others working near by, develop silicosis very rapidly and are predisposed thereby to tuberculosis and other respiratory infections.

GRANITE CUTTERS

This is a single industry, but a hazardous one from the standpoint of pneumoconiosis. The effects of granite dust inhalation have been most carefully studied from the roentgenologic standpoint by Jarvis[11] at Barre, Vermont, which he states is the largest granite center in the world. Our personal experience in this industry included only 5 cases examined, but their roentgenograms show the essential features which we have seen in the films of Jarvis' cases (Fig. 14). Granite in the Barre district, according to Jarvis[10] contains 69.56 per cent of silica, and most of the dust arises from the finishing of the stone by the pneumatic tool. Statistics quoted from him[9] on the age period will serve to show the early incidence of lung changes and advanced silicosis and to indicate the relative danger to the workers:

Earliest first stage findings under 1 year at work and greatest number: 10 to 20 years.

Earliest second stage findings 2 to 4 years at work and greatest number: 20 to 40 years.

Earliest third stage findings 10 to 14 years at work and greatest number: 30 to 40 years.

These occupation period statistics do not differ greatly from our own of advanced silicosis in hard coal miners, but a relatively much greater number of the granite workers are exposed to silica-bearing dust than is the case with anthracite miners in general. Jarvis' statistics are possibly not so bad as those among some of the hard rock miners, but they are much worse than among potters. The frequent incidence of tuberculosis has been referred to.

Jarvis[11] calls attention to an interesting observation which we have not found elsewhere and have not noted personally and which must be peculiar to the granite cutter. He describes in the lungs of many workers an area of increased lung change below the left clavicle and at the right base. He ascribes this to holding the pneumatic tool in such a manner that the right upper chest is compressed by the right arm and elbow and the left lower chest by the left elbow and forearm, thus limiting costal expansion in these two areas and permitting more dust to go to the opposite lung areas, which showed the greater changes.

POTTERS

Potters have long been known as a group of workers in whom incapacitating lung conditions are prone to develop in time and have been regarded as more or less subject to tuberculosis. A roentgen examination of the chests of a group of these workers who have been so occupied for a long period of years will readily demonstrate the correctness of the first of these two opinions, at least. As a matter of fact, they show as advanced silicotic changes as workers in any other occupation which exposes to silica dust inhalation, but the development of these changes requires a much longer period of time than in many other occupations such as hard rock miners, fire-brick workers and metal grinders. This industry is somewhat different from many of the others in that the workers

occupation and the longest time at work and remaining in this stage was thirty-five years (Fig. 16). There was a great variation in the occupation period in this stage. The earliest second stage changes were found in a kiln man after three years' occupation, and the longest occupation examined was forty-two years (Figs. 15 and 18). These statistics are, of course, not exact, as they do not indicate when the earliest evidences of the stages started; they represent records of only positive cases and represent individuals picked up conveniently for examination or those

FIG. 16. First stage pneumoconiosis in a potter of thirty-five years' occupation. Male, aged fifty-seven. This case is in marked contrast to the one shown in Figure 15. The hilum shadows are greatly increased in extent and the adjacent trunk shadows thickened. The right diaphragm was found adherent, but from other causes. Clinically the man had a large heart, and had complained of dyspnea and swelling of the feet and ankles for two months. Passive congestion might account for all the appearances, which may be practically identical in both passive congestion and the first stage of silicosis.

among second stage cases was forty years. The average occupation period was twenty-six years when examined (Fig. 17). The earliest third stage case was twenty-four years' and the oldest sixty-seven years' occupation, and the average when who had drifted into the hospital or our office for examinations for other conditions. Every potter examined showed some changes, however. We do not know the tuberculosis records, as we had no check on physical findings in most instances.

are in the open in buildings and the hazard is, in a great measure, more readily lessened. There are several ways in which the dust may be produced. For the most part, pieces of clay are dropped on the floor or benches and dry and become pulverized and the dust is kicked up. One can see how this might in a measure be avoided. The grinding of the finished of clay, flint and Cornish stone having a free silica content of 10 to 25 per cent, and that it takes twenty years to produce much change in the lungs. Landis[43] regards the pottery industry a good risk with proper care. He states that the effects on the lungs are most noticeable after forty years at work, and few workers remain at their trade after the age of

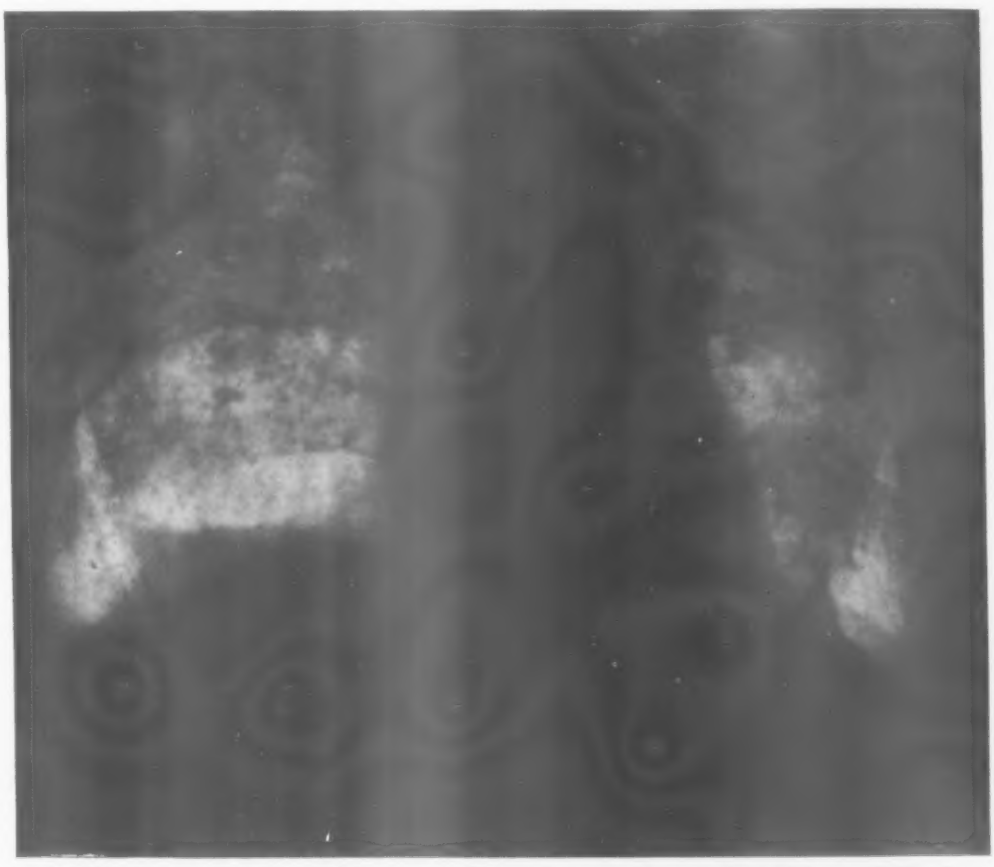

FIG. 15. Third stage pneumoconiosis in a potter of forty-five years' occupation. Male, aged fifty-five. There is a diffuse fibrosis throughout both lungs, with areas of massive fibrosis. Some of the nodular fibrosis can still be seen. The diaphragms are deformed and were almost fixed. The mediastinal structures are distorted. This man was incapacitated for work. Repeated sputum examinations were negative.

product is another source of dust. The kiln rooms and kilns are very prolific sources of dust, and no doubt more difficult to control. Here the fire brick may play an additional part.

The percentage of silica in the material used is not very high, hence the longer period of time required to produce advanced silicosis. Nicholson[50] states that the potters in England work with a mixture sixty, being incapacitated by pneumoconiosis or superimposed tuberculosis.

We have examined 58 potters showing definite evidences of pneumoconiosis. Nine showed first stage changes, 28 second stage and 21 third stage appearances. Thirty-eight of these were referred to in the report of our work with Miller and Landis in 1918.[1] The earliest evidence of first stage changes was after three years'

METAL GRINDERS AND METAL DUSTS

The grinding of metals, especially tools and parts of bearings, seems to be an industry which, in our experience, is capable of producing early and rapidly developing evidence of pneumoconiosis. We have examined 24 of these workers, earliest second stage changes were found after one year's occupation and another man showed very advanced changes after three and a half years at work. The longest occupation among second stage cases was twelve years. The average occupation period was seven years when examined. One of the third stage cases had been at

FIG. 17. Second stage pnemoconiosis in a potter of twenty years' occupation. Male, aged forty-one. There is a diffuse fine nodular fibrosis in both lungs, somewhat more marked on the right side. First stage changes are still in evidence. This man was not incapacitated because of his chest condition, but had an acute polyarthritis.

and 10 showed first stage changes, 12 second stage and 2 third stage appearances. The earliest first stage changes were found in a man after ten months' occupation, and the longest time at work and remaining in this stage was thirty-seven years. The work five years and the second case showed extreme changes with massive consolidation after eight years' occupation (Fig. 1). Of course the exact occupation is a very important factor in the pneumoconiosis in these cases.